EFFECTS OF LONG DURATION NOISE EXPOSURE ON HEARING AND HEALTH

ADVISORY GROUP FOR AERSOPACE RESEARCH AND DEVELOPMENT

PREPARED FOR
NORTH ATLANTIC TREATY ORGANIZATION

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15. Abstract

There can be no doubt that noise exposures of durations greater than eight hours present a hazard to the hearing of air crews flying noisy aircraft and, particularly, for those more susceptible crew members. We were shown today that there are wide individual differences in human response to high-level, long-duration noise. The effects of long-duration exposures on performance and health are less clear. Monitoring audiometry and ear protection is certainly indicated for preservation of hearing and perhaps, for insuring adequate performance and ultimate health of air crews flying long-duration missions. It is of additional importance that noise reduction at the source be accomplished wherever possible for safeguarding the hearing and health of those who live around NATO airports.

Collection of data on incidence of stress-induced pathologies such as ulcers or emotional disorders for those exposed to long-duration noise, as compared to non-noise exposed might be worthwhile in order to resolve the question of whether or not health is affected. It is therefore recommended first that flight crews exposed to such long durations of noise be monitored both audiometrically and for abnormal incidence of cardiovascular disease, ulcers, and other psychosomatic complaints and that secondly, if possible, a study of an appropriate laboratory animal might be instituted over the next several years which could resolve perhaps the important problem of whether or not pathology can be induced because of long-duration noise exposure to the moderate levels of noise that occur in aircraft cockpits.

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EFFECTS OF LONG DURATION NOISE EXPOSURE ON HEARING AND HEALTH

Edited by

Dr Milton A.Whitcomb
National Academy of Sciences
Washington, D.C. 20418, USA

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SUMMARY

There can be no doubt that noise exposures of durations greater than eight hours present a hazard to the hearing of air crews flying noisy aircraft and, particularly, for those more susceptible crew members. We were shown today that there are wide individual differences in human response to high-level long-duration noise. The effects of long-duration exposures on performance and health are less clear. Monitoring audiometry and ear protection is certainly indicated for preservation of hearing and, perhaps, for insuring adequate performance and ultimate health of air crews flying long-duration missions. It is of additional importance that noise reduction at the source be accomplished wherever possible for safeguarding the hearing and health of those who live around NATO airports.

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INTRODUCTORY REMARKS

An outstanding group of scientists has accepted the invitation of the Aerospace Medical Panel of the Advisory Group for Aerospace Research and Development of the North Atlantic Treaty Organization to present papers concerning a topic of great interest to the panel. It would be well, first of all, to discuss the title. One could either define a long-duration noise exposure as one that persists for more than eight hours per day and, therefore, permits less than a sixteen hour recovery period, or as those exposures that extend over years of time, or as some combination of each. The first of these, exposures of more than eight hours, is perhaps the most interesting and the one on which we will have data presented in this report. As exposures extend toward twelve hours, recovery periods are similarly reduced toward twelve hours. It may be that the recovery period is a critical factor for the auditory system, not only in terms of the effect on hearing, but also the effect on performance, on health, or on any of a host of other parameters. One could define the effect of noise exposure on hearing as one that produces a temporary insensitivity to sound (temporary threshold shift), or as a permanent insensitivity to sound (permanent threshold shift). Finally, one could define health as does the constitution of the World Health Organization, in which it is stated "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity". This is a laudable definition of health since all too long health has been couched in negative terms only such as absence of pathology. It is refreshing to see health defined positively in terms of a need to feel good. Yet one should be careful to be very clear when one talks about the effects of noise on health in these proceedings to state whether or not one is discussing pathology and tissue damage or whether one is talking about the absence of a state of mental well-being. For example, annoyance could be considered a health effect in that noise does annoy us and therefore affects our health. As long as the reader of these proceedings knows that this is what is meant, there is no problem. However, it would be of grave concern if an uninformed person would believe that moderate levels of noise damage tissue when all the author intended was that moderate levels of noise cause annoyance. One could become unduly alarmed. Certainly this is not desirable.

The outline for the papers contained in this publication very briefly is as follows. First there will be a set of papers dealing with the effects of long-duration noise exposure on the hearing of man as well as animals. These effects will be both those which are transient in that the effects are recoverable to initial base line audiograms of either man or animals and secondly, those which are permanent in that the effects do not recover to initial base line audiograms, even after several days following exposure of man or animals to such noise. Another set of papers will deal with the effects of long duration noise exposure upon the physiology of man or animals and, again, there will be papers concerned with transient physiological effects on man and animals followed by papers concerned with permanent physiological effects on man or animals. Finally, there will be a set of papers concerned with the effects of long-duration noise exposure upon performance and upon health in general.

There can be no doubt that hearing is affected by long-duration noise exposure for some pilots, namely those who are most susceptible; flying some airplanes, namely those which are the noisiest, for some durations, namely those extending beyond eight hours per day for several years, and for some number of years of exposure, at some criterion level of hearing loss. For example, if the criterion level of hearing loss is zero audiometric change, even at 4,000 Hz, there is no double that some loss will occur. Whether loss occurs at other criterion levels such as 25 dB averaged across 500, 1000 and 2000 Hz audiometrically, or still others yet to be named, is less certain. However, it is still likely that for the NATO situation, some pilots, flying some airplanes, for some durations per day, for some number of years, will exceed even these lenient criteria for hearing loss. The question to be faced in this symposium is: How bad is the problem tody as judged from research data applied to NATO missions? If there is a problem, what can be done about it? Can it be resolved by scheduling of personnel? Can it be resolved by wearing of personal protective equipment such as ear plugs and ear muffs? Can it be resolved only by the control of the noise at the source? Is health affected in terms of pathology that would occur over a long period of time and not simply transient cardiovascular changes that occur during the duration of the noise exposure itself? This is, of course, an open question, but one that cannot be ignored because if there are long term pathological effects, one is obliged to understand their etiology and attempt to do everything one can to eliminate them. Finally, is performance affected? If so, it could effect the efficiency of NATO pilots.

It would be important during the discussion periods to relate questions and answers to Aerospace Medical Panel concerns.

Dr Milton A Whitcomb
Committee on Hearing, Bioacoustics
and Biomechanics
National Academy of Sciences
Washington, D.C., USA

CONTENTS

	Page
AEROSPACE MEDICAL PANEL	iii
SUMMARY	iv
INTRODUCTORY REMARKS	v
	Reference
MODE OF COCHLEAR DAMAGE BY EXCESSIVE NOISE – AN OVERVIEW by D.J.Lim and W.Melnick	C-1
TTS IN MAN FROM A 24-HOUR EXPOSURE TO AN OCTAVE BAND OF NOISE CENTERED AT 4 KHZ by W.Melnick	C-2
PROTECTIVE EFFECTS IN MEN OF BRAIN CORTEX GANGLIOSIDES ON THE HEARING LOSS INDUCED BY HIGH LEVELS OF NOISE by G.Maniero and G.A.Molinari	C-3
STUDIES OF ASYMPTOTIC TTS by W.D.Ward	C-4
ASYMPTOTIC BEHAVIOR OF TEMPORARY THRESHOLD SHIFT DURING EXPOSURE TO LONG DURATION NOISES by D.L.Johnson, C.W.Nixon and M.R.Stephenson	C-5
THE INCIDENCE OF TEMPORARY AND PERMANENT HEARING LOSS AMONG AIRCREWS EXPOSED TO LONG-DURATION NOISE IN MARITIME PATROL AIRCRAFT by S.E.Forshaw	C-6
PSYCHO-PHYSICAL PERFORMANCE OF AIR FORCE TECHNICIANS AFTER LONG DURATION NOISE EXPOSURE by C.A.Ramacci and P.Rota	C-7
THE EFFECTS OF EAR PROTECTORS ON SOME AUTONOMIC RESPONSES TO AIRCRAFT – AND IMPULSIVE NOISE by G.R.Froehlich (Paper not presented at the meeting)	C-8
INFLUENCE OF THE NOISE ON CATECHOLAMINE EXCRETION by G.Paolucci	C-9
EFFECTS OF NOISE EXPOSURE by R.W.Cantrell	C-10
PHYSIOLOGICAL EFFECTS OF NOISE by R.W.Cantrell	C-11
AN INVESTIGATION OF AIRCRAFT VOICE COMMUNICATION SYSTEMS AS SOURCES OF INSIDIOUS LONG-TERM ACOUSTIC HAZARDS by R.T.Camp, B.T.Mozo and J.H.Patterson	C12
PHYSIOLOGICAL RESPONSES DUE TO NOISE IN INHABITANTS AROUND MUNICH AIRPORT by G.Jansen	C 13
CONCLUDING REMARKS	

MODE OF COCHLEAR DAMAGE BY EXCESSIVE NOISE
--AN OVERVIEW--

David J. Lim, M.D., and William Melnick, Ph.D.
Otological Research Laboratories and Auditory Research Laboratories
Department of Otolaryngology
The Ohio State University College of Medicine
456 Clinic Drive
Columbus, Ohio 43210, U.S.A.

Summary

The mode of cochlear damage caused by excessive noise is generally believed to be physical or metabolic stress exerted on the sensory cells. Evidence to support both mechanisms is overwhelming. Injury to the sensory cells may be direct physical or metabolic effects.[1] Subsequent added injury can be brought about by the chemical or metabolic alteration in the surrounding medium. Besides apparent mechanical damage inflicted on the organ of Corti by the acoustic hyperstimulation, the evidence of metabolic damage to the sensory cells is subtle. The e subtle changes include: 1) proliferation and vacuolization of endoplasmic reticulum in sensory cells, 2) swelling of mitochondria in both sensory cells and afferent nerve endings, 3) morphological alteration of stereocilia, and 4) swelling and degeneration of stria vascularis. These findings would imply that the high-energy-yielding enzyme systems are rendered inoperative in these cells, resulting in cell degeneration.

Introduction

It is well established that damage of the cochlea is produced by various types of noise, such as impulsive noise, intermittent or continuous noise, and low frequency or high frequency noise. Regardless of the type of noise, the basic mechanism involved in acoustic trauma is due to physical and/or physicochemical (metabolic) stress exerted on the maximally stimulated sensory organ. The end result is sensory cell damage or even cell destruction which accounts for the resultant hearing loss, which can be either temporary (TTS) or permanent (PTS) depending on the extent of injury. The purpose of this paper is to review various proposed modes of cochlear damage resulting from excessive noise.

Relevant Anatomy and Physiology

The cochlea in mammals resembles a snail shell and is divided by membranes into three major fluid-filled compartments, known as scalae vestibuli, tympani and media. The former two spaces contain perilymph and are interconnected with each other and also open to the cerebrospinal fluid via the cochlear aqueduct. The scala media is filled with endolymph in the self-contained cochlear duct. The perilymph is high in Na^+ (150 mEq/l) but low in K^+ (4.8 mEq/l), whereas the endolymph is low in Na^+ (2.5 mEq/l) but high in K^+ (150 mEq/l). Besides these fluids, cortilymph, which bathes the Corti's tunnel (Corti's tunnel and Nuel's space), is now accepted as a third lymph.[2] The chemical characteristics of cortilymph are thought to be similar to perilymph, according to Rauch.[3] There are several studies[4] using tracer particles which support the concept that this cortilymph communicates freely with perilymph (Fig 1). These findings would imply that the sensory cells are bathed with fluids high in sodium ions (cortilymph) and that the oxygen and supply may come from the perilymph of the scala tympani rather than endolymph.[5] They further inferred that the spiral vessel is directly responsible for viability of the organ of Corti, and that the cortilymph receives its oxygen supply from the spiral vessels.

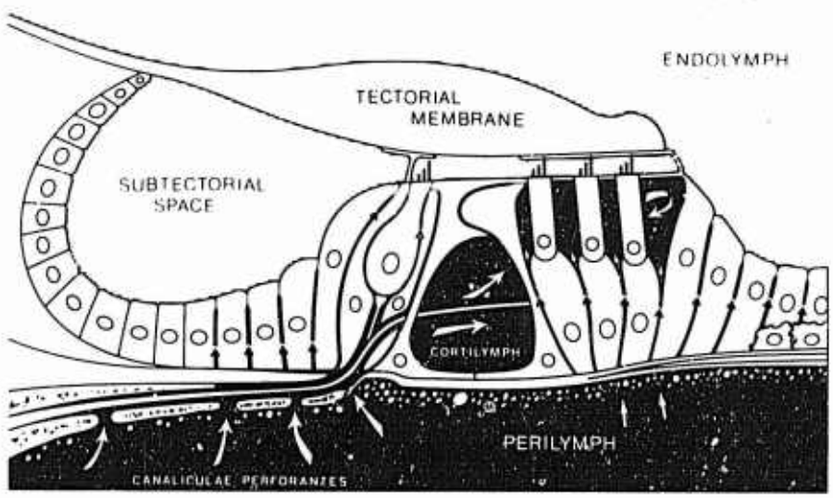

Fig 1. Artist's conception of communicating routes between cortilymph and perilymph in the scala tympani. Arrows indicate flow of inner ear fluid. Reproduced from Lim.[4]

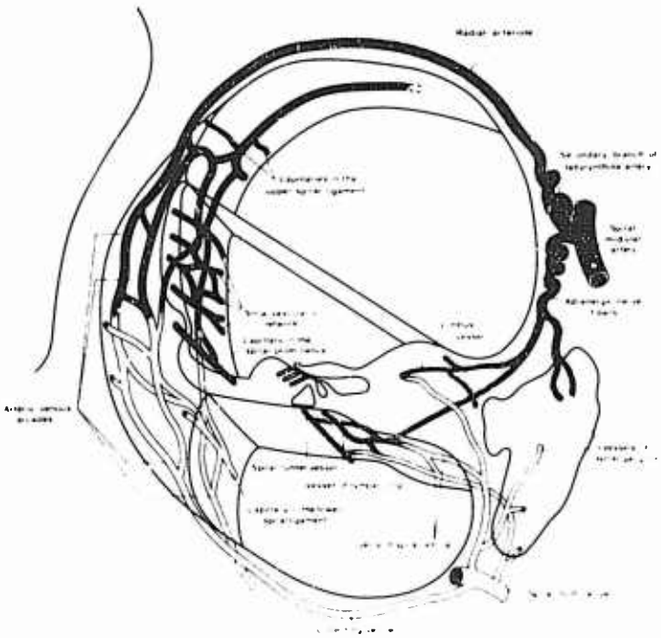

Fig 2.

A schematic diagram of blood supply to the cochlea. Modified from Pearlman and Kimura. (Ann Otol Rhinol Laryng, 64:1179, 1955)

The blood supply to the cochlea is well established, as illustrated in the schematic drawing (Fig 2). It can be divided into five major parts: 1) spiral vessels underneath the organ of Corti, 2) limbus vessel, 3) arterio-venous arcade in spiral ligament, 4) strial network, 5) spiral prominence vessel. Due to their proximity to the organ of Corti, and on the basis of his experiments, Lawrence[6] considers the spiral vessels to be the primary source of nutrients and oxygen supply to the organ of Corti. Autonomic (adrenergic) nerve fibers are known to accompany the labyrinthine arteries in the modiolus and are suggested to originate in the stellate ganglion.[7] It is generally agreed that adrenergic fibers have not been found in the stria vascularis. Wersall[8] reported the presence of pericapillary adrenergic nerve fibers in spiral vessels; however, Spoendlin[7] maintains that unmyelinated nerve fibers do not accompany spiral vessels. He also reported the presence of adrenergic nerve fibers unrelated to the spiral vessel in the osseous spiral lamina, which is thought to originate in the superior cervical ganglion. It is conceivable that the former pericapillary autonomic nerve fibers may play a role in normal homeostasis of inner ear fluid. However, it is not clear whether the latter independent adrenergic fibers are involved with the mechanism of acoustic trauma.

The endoplasmic reticular system of the outer hair cells is formed by subsurface cistern, Hensen's body, and subsynaptic cistern (Fig 3). Although its specific function is not yet wholly understood, Lim and Melnick[9] postulated a specialized function of the ER system in sensory excitation of the cell similar to the sarcoplasmic reticulum. They further showed the alteration of the ER system by excessive auditory stimulation.

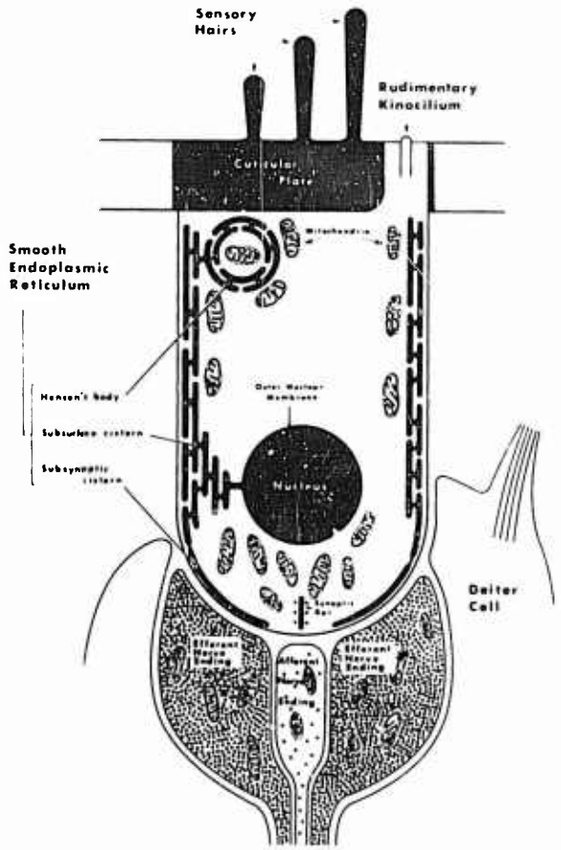

Fig 3.

A schematic diagram of endoplasmic reticular system of outer hair cells. Reproduced from Lim and Melnick.[9]

Mode of Mechanical Damage

It is well established that excessive noise can cause a detachment of the organ of Corti and a tear in the basilar and Reissner's membranes, as shown in Fig 4. According to Schuknecht and Tonndorf,[10] the stresses developed within the cochlear partition by sound waves produce direct mechanical damage to the sensory cells. The basilar membrane has a width and stiffness gradient along the length of the cochlea. It is wider at the apex but narrower and stiffer at the base. These physical differences provide a mechanical basis for frequency analysis in the cochlea. Thus, low frequency sound produces maximum cochlear effects in the apical regions, while high frequencies affect only those structures in the base of the cochlea. Since the low frequency sound creates involvement of a greater portion of the basilar membrane than the high frequency sound, the extent of sensory organ damage may be widespread as a consequence of low frequency sound stimulation.

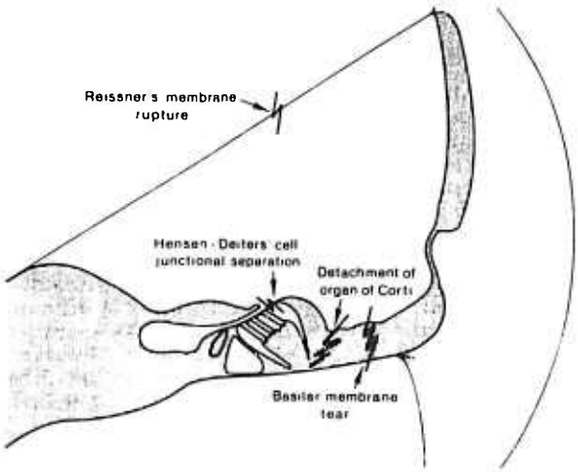

Fig 4. A diagram of the cochlea indicates area most commonly damaged by mechanical force created by acoustic stimulus.

How the sensory cells, particularly the outer hair cells, are preferentially damaged by acoustic over-stimulation is not clearly understood. Several hypotheses as to how mechanical force can cause sensory cell injury have been proposed: 1) Violent fluid motion in the cochlea partition can rupture Reissner's membrane, mixing endolymph with perilymph. This contaminated endolymph can cause damage to the sensory cells, as shown in Lim's study[4] with trace substance. 2) Violent basilar membrane motion can also cause disruption of the reticular membrane of the organ of Corti, as reported by Beagley[11] and Bohne.[12] This disruption can cause mixing of endolymph with cortilymph, resulting in the poisoning of the sensory cells. Bohne noted that the damaged sensory cells created small lesions in the reticular membrane as a result of acoustic trauma. These openings would permit intermixing cortilymph with endolymph and would cause spreading of damage to the sensory cells that were not initially injured by mechanical force. 3) Violent fluid motion in the cochlear partition can directly damage the sensory cells by detaching the organ of Corti from the basilar membrane or tearing up the basilar membrane. 4) Separation of the tectorial membrane from the sensory cilia as the result of violent fluid motion can cause threshold shift.

Mode of Metabolic Damage

Besides the obvious mechanical damage just mentioned, there are more subtle forms of morphologic change or damage in the organ of Corti or stria vascularis. This injury is considered to be the result of metabolic changes occurring during hyperstimulation. The mechanism involved in metabolic stress by acoustic overstimulation is not wholly understood. However, it has been suggested that the sensory cell damage is due to metabolic exhaustion, which includes enzyme and energy reserve depletion and reduction of oxygen and nutrient supply. Since the sensory cells are thought to be more metabolically active than the supporting cells, Lim and Melnick[9] suggested that the sensory cells are more vulnerable under stress. The damage caused by metabolic stress in the cochlea is subtle and often obscured by coexisting mechanical damage.

First, Hawkins[13] has shown that acoustic hyperstimulation causes disruption of blood circulation in the spiral vessel and the strial vessels by the swelling of endothelial cells. He further suggested that the sensory cell degeneration is secondary to the strial degeneration. Our own morphological data show marked capillary vasoconstriction in the stria vascularis, but in the spiral vessels the constriction was not so remarkable. Extensive pathological changes in the stria vascularis, particularly involving the intermediate cells, are considered to be the results of metabolic changes caused by capillary injury due to noise (Fig 5). According to Duvall et al,[14] these changes are confined to the intermediary cells, and are reversible, meaning the degenerated intermediate cells have reappeared as hearing was recovered. The mechanism involved in this vasoconstriction by the sound is not yet known. Remarkable endothelial cell swelling can be caused by direct physical stimuli, but also it can be speculated that the unmyelinated nerve fibers accompanying the cochlear vessels are autonomic fibers which are responsible for this vasoconstriction.

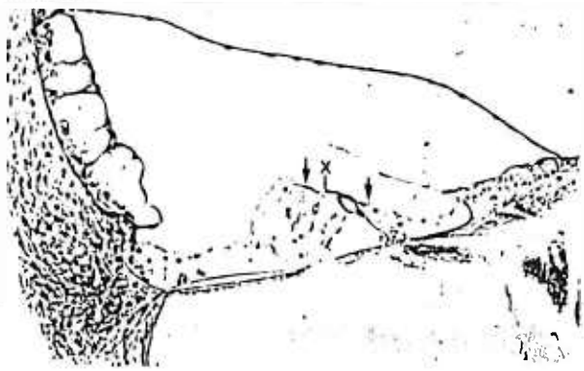

Fig 5.

A phase contrast photomicrograph shows extensive swelling of the stria vascularis and degenerated two outer hair cells indicated with X. The third outer hair cell and inner hair cell appear intact (arrows). Animal was exposed with 140 dB wide band noise exposure for 5 minutes and sacrificed 24 hours later. (144X)

Second, acoustic hyperstimulation caused a proliferation and eventual vesiculation of the endoplasmic reticular (ER) system, as well as the swelling of the mitochondria that are attached to the ER in the sensory cells. As pathology progressed, the vesiculation extended not only in ER membranes, but also in the nuclear membrane and subsynaptic cistern (Fig 6-A). The efferent nerve endings which are attached to degenerating sensory cells appear still intact, while the afferent nerve endings of the same sensory cells showed distinct mitochondrial swelling. These findings appear to support the concept that acoustic trauma causes disruption of enzyme systems which are essential for protein, glucose and lipid synthesis, and these enzyme systems reside in ER and mitochondria. How this alleged disruption comes about is not wholly understood, but it is suspected to be the result of anoxia in the cells caused by acoustic hyperstimulation. Misrahy et al[15] found that endolymph oxygen tension initially increases following sound exposure and then decreases rapidly and markedly. The original level is restored only after a prolonged period of time, which is in proportion to the intensity and the duration of exposure. Vosteen[16] showed that a respiratory enzyme (succinic dehydrogenase) was greatly reduced in the outer sensory cells but not in the inner hair cells by acoustic over-stimulation and hypoxidosis. He further speculated that the concommitant oxygen deficit within the hair cells is made worse by increased oxygen demand, due to the heavy load upon the sensory receptors. Consequently, after an acoustic overload, the sensory cells ought to show the same structural changes as occur in other cells in hypoxia. Indeed, our earlier report showed that some sensory cells had signs of degeneration while adjacent cells did not (Fig 6-A), supporting Vosteen's notion. The cells that are degenerated are in a greater active metabolic state than the ones that are not degenerated by the same stimulus. Furthermore, Ishii et al[17] demonstrated the glycogen in the outer hair cells diminished appreciably by prolonged sound exposure. This finding would imply that the energy needed for sensory cell function is obtained from glycolysis. Therefore, when the main source of energy is depleted, the sensory cells can no longer function, implicating metabolic depletion as a cause of hearing loss from noise exposure.

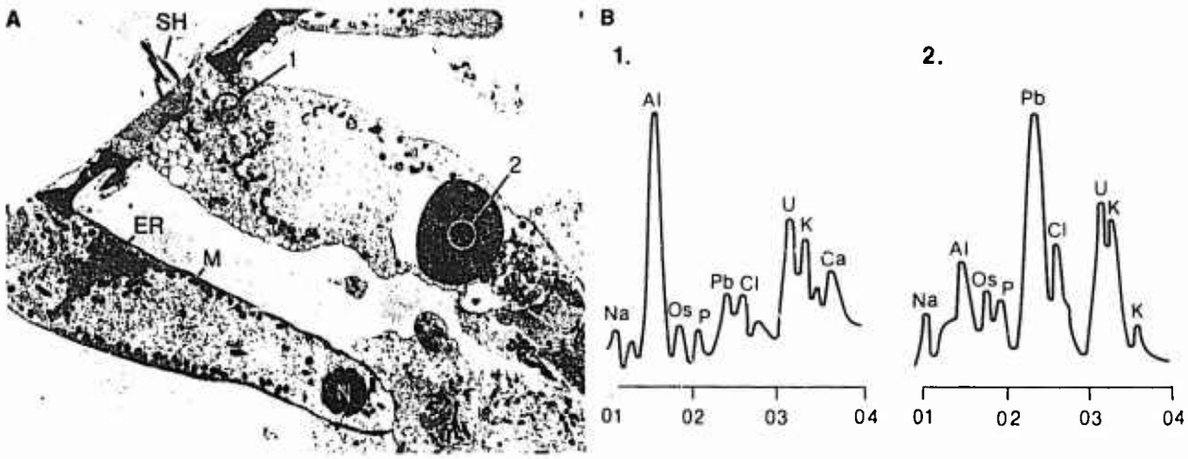

Fig 6 A. Electron micrograph shows vacuolated and vesiculated outer hair cells at the second turn. SH - sensory hairs, ER - endoplasmic reticulum, M - mitochondria, N - nucleus. Numbered areas are examined with X-ray analyzer. (300-600 Hz, 117 dB SPL, 4-hour exposure, 10 days recovery.) Reproduced from Lim and Melnick.[9]

B. X-ray analysis of dark bodies (1) and nucleus (2) are shown. Remarkable calcium peak is seen in lysosomal bodies, but not in nucleus. Modified from Lim's original data.[18]

Lim[18] earlier demonstrated elevated calcium in the dark granules (lysosomes) in the sensory cells that were subjected to acoustic hyperstimulation (Fig 6-B). This finding may support the possibility that calcium ions in the cytoplasm can be bound to and released from the ER as a function of sensory cells, and when the ER system becomes injured by acoustic trauma, the unbound surplus calcium can be accumulated in the lysosomes. However, this concept is highly speculative.

Third, there have been reports that fusion of sensory hairs is often noted in animals that were exposed to intense noise. Similar changes were also noted in cochlea sensory hairs damaged by lasers, as well as vestibular sensory hairs damaged by ototoxic drugs. The mechanism by which these cilia fuse is not known, but it has been suggested that the membrane electrostatic properties[19] or permeability of the stereocilia have changed.[20] Whatever the reason may be for this fusion, it can be suggested that this fusion is not specific for acoustic trauma. Perhaps this pathology represents general degenerative changes involving sensory cells, most likely due to protein and/or lipid denaturation. Perhaps the deformed cuticular plate in the sensory cell which results from acoustic trauma observed by Lim and Melnick[8] can also be interpreted as caused by the disruption of protein metabolism, as suggested earlier by Vinnikov and Titova[21] and others.

Fourth, an unexpected observation was the apparent displacement of the basal body (rudimentary kinocilium) in both inner and outer hair cells, following acoustic stimulation (Fig 7). Whether this displacement can cause functional impairment of the sensory cells, or they can be restored spontaneously as hearing recovers, cannot be determined. If one subscribes to the concept that the basal body has functional significance in sensorineural excitation, then one can speculate that the displacement of the basal body would impair the function of sensory cells, causing TTS. On the other hand, similar observations have been made in a few presumably normal sensory cells, which raises a question of validity of this speculation. Furthermore, these findings raise questions regarding the assumption that the basal body is a fixed structure. The possibility that the displaced basal body is a wandering centriole, which has similar morphologic features to a basal body, cannot be ruled out.

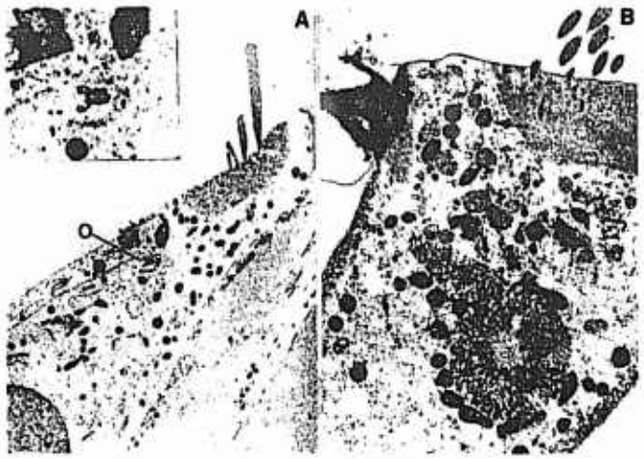

Fig 7 A. Inner hair cell in apical turn shows a displaced basal body or wandering centriole (O). (300-600 Hz, 85 dB, 72-hour exposure, no recovery) (4,000X)

B. Outer hair cell in apical turn of the cochlea shows a displaced basal body (arrow) following acoustic stimulation. (300-600 Hz, 85 dB, 4-hour exposure, no recovery) (8,000X)

Concluding Remarks

While the mode of mechanical damage inflicted on the hearing organ by excessive acoustic stimuli is better understood, the concommitant and subsequent (progressive) mode of metabolic injury to the sensory cells is poorly understood. Understanding of the latter mechanism is further retarded by our lack of knowledge concerning energy metabolism related to the hearing organ and concerning the biochemical events in the sensory cells involved in neural excitation. Several investigators attempted to avert these metabolic changes in the inner ear by giving vitamin A[22] or hydroxyzine hydrochloride,[23] but without much success. Faltynek and Vesely,[24] claimed that ATP and AMP were useful in restoring cochlear microphonics after acoustic trauma and hypoxia. They even used ATP for the treatment of sudden deafness and reported an improvement of hearing in most of their patients.[25] However, the value of this therapy has to be further documented.

It is hoped that when we understand the biochemical events leading to the acoustic trauma more clearly, then we may be able to prevent or avert further progressive damage of the inner ear by medical means.

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TTS IN MAN FROM A 24-HOUR EXPOSURE TO AN OCTAVE BAND OF NOISE CENTERED AT 4 KHZ

William Melnick, Ph.D.
The Ohio State University
Department of Otolaryngology
456 Clinic Drive
Columbus, Ohio 43210

Seven men were exposed to 24 hours of continuous noise in a sound field. The noise was an octave band centered at 4 kHz at two octave band levels, 80 and 85 dB. Hearing thresholds were measured in one ear at 11 test frequencies ranging from 250 to 10,000 Hz prior to exposure and at selected intervals during and after exposure. Temporary threshold shift (TTS) reached asymptotic levels between 8 and 12 hours of exposure. Maximum TTS occurred at 4 and 6 kHz. Asymptotic levels at the 80 dB exposure level were 9.7 dB for 4 kHz and 7.7 dB for 6 kHz. With the 85 dB noise level, these levels were 18.4 dB and 16.5 dB, respectively. Asymptotic threshold shift (ATS) could be predicted by the equation ATS = 1.75 (OBL - 75). Threshold shift for this subject group was less than would be expected from results of previous investigations.

Introduction

Information about the effects of duration of exposure to noise on the hearing sensitivity of man and lower animals has come chiefly from investigations that have used exposures lasting eight hours or less. Investigations using longer exposure times have used subhuman experimental animals, mainly the chinchilla, because of the nature of the experiment. (1,2,3,4,5) The major finding of these investigations was that threshold shifts increased for the first 24 hours and then reached a plateau, or asymptote. Asymptotic threshold shift (ATS) in the frequency region of maximum change of sensitivity increased at a rate of about 1.6 to 1.9 dB for every dB increase in the octave band level of the exposure noise above some minimum value. This minimum value was found to be 47 dB for noise centered at 4 kHz and about 65 dB for noise centered at 500 Hz. (2,3) In chinchillas, recovery from ATS is slow. Recovery could take anywhere from 3 to 30 days, depending upon the characteristics of the noise exposure. (6)

Relatively few investigations have used exposures of sufficient duration to produce asymptotic threshold shift in humans. (7,8,9,10) From these investigations, it appears that TTS grows faster for humans than for chinchillas. Asymptotic levels were achieved after 8 to 12 hours of noise exposure. Human subjects seemed less sensitive to noise than were chinchillas. Comparatively high levels were required to produce noticeable shifts in threshold sensitivity. For an octave band of noise in the region of 500 Hz, threshold shifts at asymptote in the frequency of maximum effect increased about 1.7 dB for every dB increase in level of noise above 75 dB octave band level. As with the chinchilla, recovery from asymptotic threshold shift is slow, requiring anywhere from 1 to 6 days. (6)

Since 1971, a series of experiments have been run in our laboratory which have investigated the growth of and recovery from TTS in humans as a result of prolonged exposure. The first series of experiments used 16 hours of exposure to an octave band 300 to 600 Hz at band levels of 80, 85, 90, and 95 dB. (9) The 16-hour exposure period was not long enough to establish clearly that asymptotic levels of threshold shift had been reached. A subsequent experiment increased the exposure duration to 24 hours, using a 90 dB octave band level. (10) Asymptotic levels were achieved by 12 hours of exposure. The growth pattern of TTS was tri-phasic: slow development during the first two hours of exposure, a rapid increase from two to eight hours of exposure, and then plateauing by the twelfth hour. The TTS from these experiments could be predicted by the equation ATS = 1.7 (OBL - 75).

The present series of experiments have continued to use a 24-hour exposure, but have shifted the spectrum of the exposure noise to an octave band centered at 4 kHz. The human auditory system appears to be most susceptible to noise exposures in this frequency range. (11)

Procedure

Subjects: Seven men served as subjects. These men were recruited from the male inmate population of a penal institution. To be eligible for the study, a subject's hearing threshold level for both ears could not exceed 15 dB HTL at any frequency (ANSI-1969). (12)

One ear was selected arbitrarily as the test ear for each subject. Air conduction thresholds were measured at 10 test frequencies ranging from 250 to 10,000 Hz using a Bekesy-type procedure. The test signal was pulsed with a period of 500 msec, a duty cycle of 50%, and a rise-decay time of 25 msec.

Subjects tracked their own threshold for 40 seconds at each frequency. Test tones were presented in an ascending order, starting with 250 Hz. Estimates of pre-exposure threshold levels were based on the mean of ten threshold measurements made prior to the exposure period. Three separate measurements were made on three successive days with each subject. The tenth measurement was made immediately prior to the onset of the 24-hour exposure period.

Hearing threshold levels were measured at nominal intervals 1, 2, 4, 8, 12, 16, 20, and 24 hours during the exposure. The noise was interrupted one minute before the actual measurement began, and the noise resumed immediately upon completion of the measurement at all test frequencies. Threshold measurement was made following the same procedure used to obtain pre-exposure thresholds. The subject himself placed and removed earphones on signal from the experimenter.

The noise was interrupted for 3 minutes and 20 seconds for each measuring period. To offset the effects of this interruption, exposure time was increased 3 1/2 minutes for each interruption period. After the measurement made one hour into the noise exposure, each subsequent exposure period was increased by this correction factor. The correction was based on the assumption that an increase in exposure duration of 3 minutes offsets recovery which would occur during an interruption of seven minutes, and is similar to that used by Mills et al. (7)

In measuring recovery of sensitivity, subjects were tested at selected intervals following the exposure, approximately 2, 4, 8, 24, 48, and 72 hours A subject's activity was not under experimenter control during the 72-hour recovery period, but he was asked to avoid exposure to any loud sound. These subjects worked in relatively quiet job environments or were in their dormitory rooms in intervals between experimental sessions. Because of lack of total control, however, it is possible that pre-exposure levels could have been inflated, and recovery of hearing sensitivity following noise exposure could have been delayed.

Threshold measurements and noise exposure took place in the same sound isolated test room. The subject was exposed to a sound field generated by loud speakers. The noise was nominally an octave band centered at 4000 Hz. Two octave band levels were employed, 80 and 85 dB. An analysis of the exposure noise using 1/3 octave band intervals is shown in Figure 1. These measurements were taken in a standard microphone position. Since the room was not highly reverberant, the sound field could not be described as diffuse. Variations of 5 to 6 dB were measured at particular third octave bands as the microphone was moved around the room.

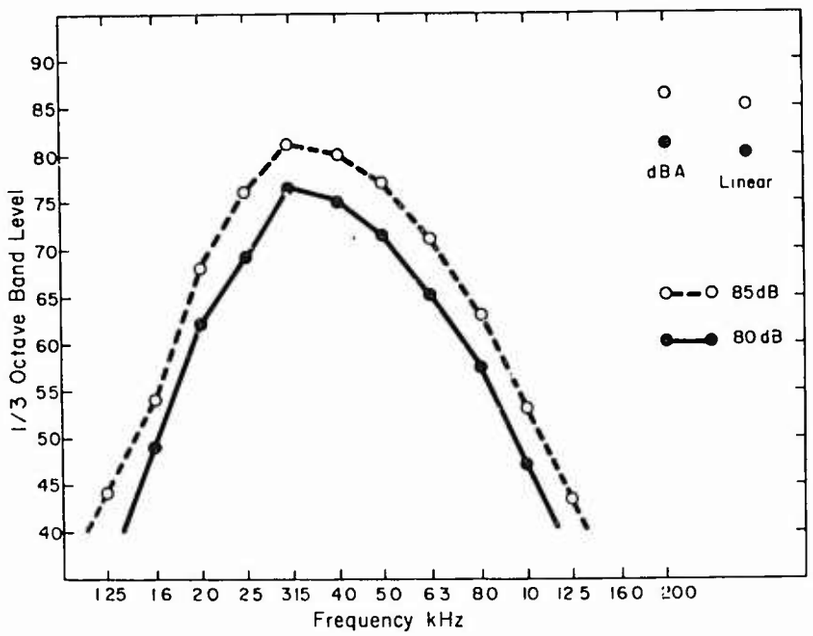

Figure 1
Spectra of exposure noises for two exposure levels as measured in one third octave bands at a standard microphone location. Measures using the A and linear weighting networks are indicated.

The pure tone test frequencies were produced by a beat-frequency oscillator which was manipulated manually by the experimenter. The test frequency was monitored by an electronic counter and was set within one per cent of the nominal frequency. A decade attenuator and a line amplifier were used to set the overall signal level. The signal was directed to a recording attenuator through an impedance matching transformer and finally to the test earphone. The attenuation rate of the recording attenuator was 4 dB per second.

Results and Discussion

The mean pre-exposure hearing threshold levels (ANSI-1969) for the seven subjects are shown in Figure 2. The bars at each test frequency indicate ± one standard deviation. These subjects showed an average of 5 dB or less hearing loss at frequencies above 1000 Hz, the frequencies important in this investigation. Showing standard deviations may be inappropriate and misleading, since the distribution of hearing levels for the subjects would not be symmetrical because of the imposed maximum of 15 dB hearing loss. Since the subjects in this experiment were males in the third, fourth, and fifth decades of life, these thresholds indicate rather good sensitivity in the experimental group.

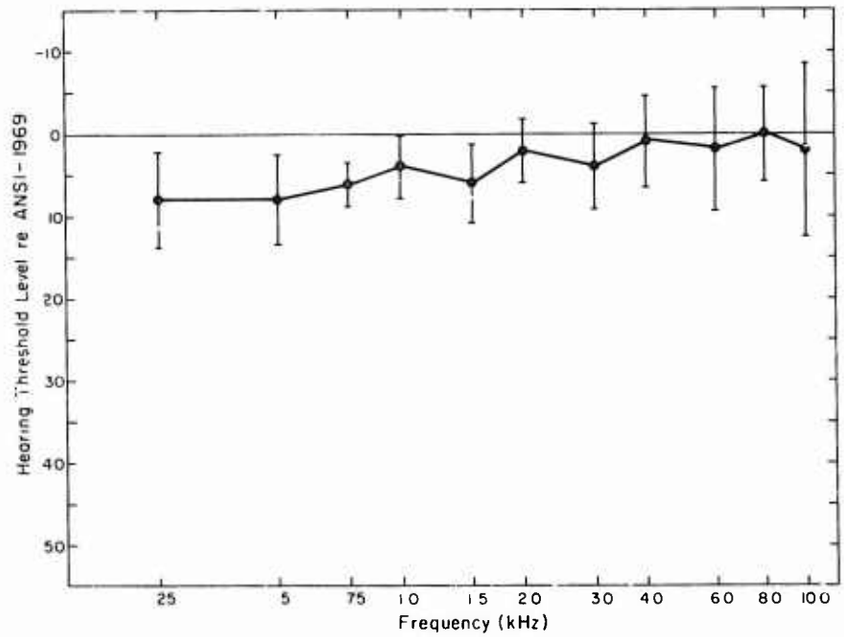

Figure 2
Mean pre-exposure hearing threshold levels (ANSI-1969) for seven subjects. Bars represent ± one standard deviation.

Temporary threshold shift was calculated by subtracting the pre-exposure hearing level for a given test frequency from threshold levels measured for the same frequency during and after the noise exposure. The threshold shifts reported have not been converted to a common time following exposure. Threshold shifts represent estimates at the times following noise interruption dictated by the experimental procedure. TTS at 3.0 kHz then would be an estimate made at 5 minutes 40 seconds or TTS 5:40; 4.0 kHz is TTS 6:00; 6 kHz, TTS 6:40; 8 kHz, TTS 7:20, etc.

The pattern of threshold shift by frequency is indicated in Figure 3. These data points represent asymptotic levels, and were derived by averaging the measures made at 8, 16, 20, and 24 hours of exposure for each of the exposure levels 80 and 85 dB. Maximum threshold shift was seen at the frequencies 4000 and 6000 Hz. There were measureable threshold changes at 3000 and 8000 Hz, but no measureable effects at the other test frequencies.

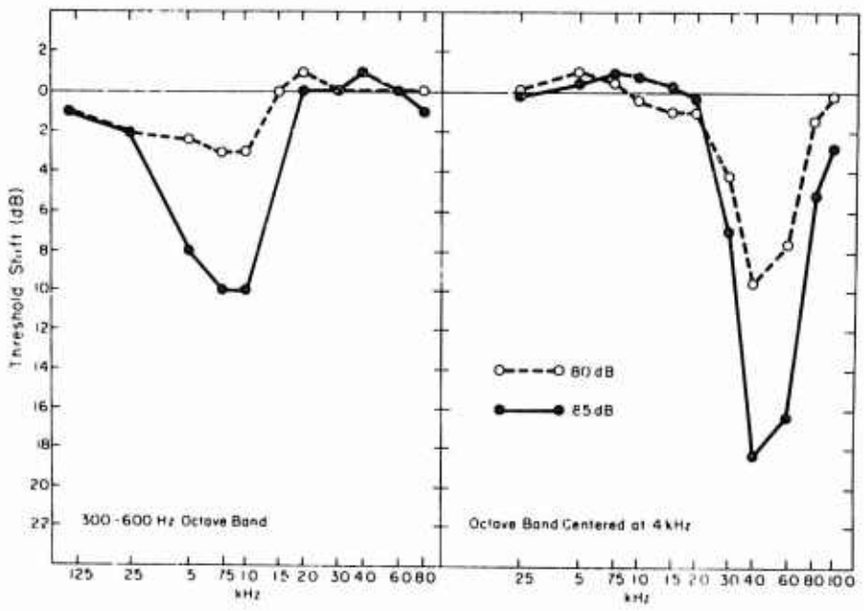

Figure 3
Asymptotic threshold shifts (ATS) by test frequency for 80 and 85 dB noise exposures. Left graph contains data from a previous study (9) using a 16-hour exposure to a 300-600 Hz octave band of noise. Right graph shows results from the present study. ATS was calculated from average thresholds

measured over 8 to 24 hours of exposure for present experiment and 8 to 16 hours in the earlier study.

The left hand graph in Figure 3 gives comparative data obtained from our earlier experiments using a 300 to 600 Hz octave band of noise at 80 and 85 dB levels. (9) In the earlier studies, however, the exposure was terminated at 16 hours, so the asymptote represents an average of the measurements made at 8, 12, and 16 hours of exposure. From these data, it would seem that we obtained less maximum threshold shift using the lower frequency exposure, and the effect seemed broader than with the higher frequency noise. A direct comparison of these results is not possible, however, since the same subjects were not used in both series of experiments. As a matter of fact, the subjects who were exposed to the lower frequency noise showed threshold hearing levels of about 10 dB in the frequency range of importance, 250, 500 and 750 Hz. If this hearing loss were taken into account, then the magnitude of the peak asymptotic threshold shift for the two different exposure noises would be in the same range.

The development of and the recovery from TTS produced by the 80 dB octave band level is shown in Figure 4. TTS is graphed for the four frequencies which showed any appreciable threshold shift. The asymptote for these frequencies apparently was reached sometime between 8 and 12 hours of exposure. The maximum threshold shift occurred at 4000 cycles, and when the threshold shift is averaged over the 8 to 24 hour measurement periods, the magnitude of the group ATS for 4 kHz was 9.7 dB with a range of 5 to 21 dB. For 6 kHz, the asymptote was 7.7 dB with a range of 4 to 13 dB. Looking at these results, it would be difficult to say if any significant threshold shift occurred at 8000 Hz. The pattern of recovery indicates that even though the magnitude of TTS was not great, it took 24 hours before threshold approximated pre-exposure levels.

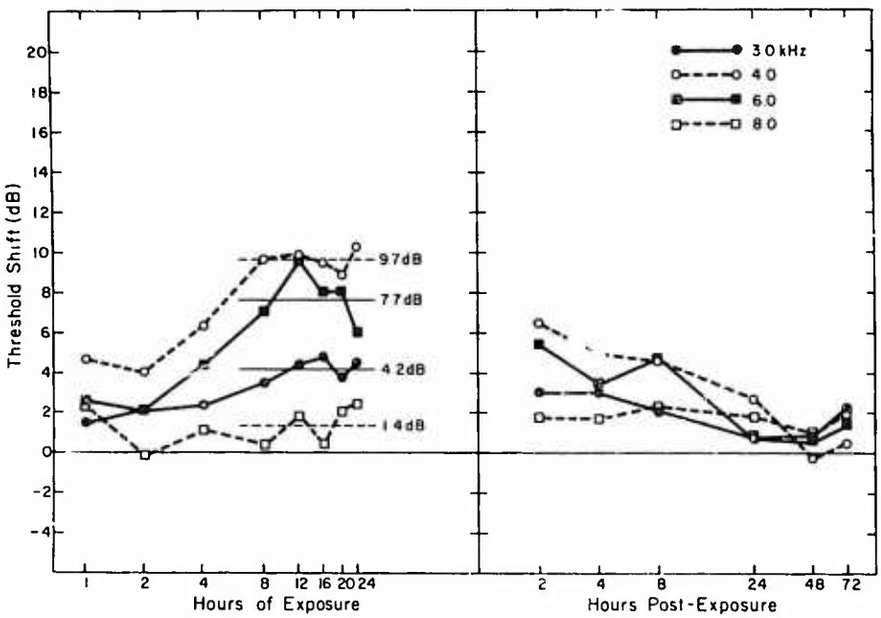

Figure 4
Development of TTS (left graph) and recovery from TTS (right graph) for test frequencies 3, 4, 6 and 8 kHz, resulting from exposure to an octave band of noise centered at 4 kHz at 80 dB octave band level. Data points are the mean for seven subjects.

Figure 5 displays the development and recovery of threshold shift from the 85 dB exposure level. The asymptotic levels were calculated in the same fashion as for the 80 dB exposure, and were 18.4 dB for 4000 Hz and 16.5 dB for 6 kHz for the group. The asymptotic threshold for individual subjects ranged from 8 to 30 dB at 4 kHz and 7 to 33 dB for 6 kHz. The separation in the magnitude of TTS for these frequencies showing maximum shift is much greater at this exposure level, indicating a more rapid growth of TTS with increase of intensity at the most affected frequencies 4 and 6 kHz, with relatively slow growth in the fringe frequencies, 3 and 8 kHz. The asymptote for 3 kHz was calculated to be 7 dB, while the level for 8 kHz was 5.3 dB.

Again, recovery is relatively slow when the small magnitude of threshold shift is considered. Although threshold recovered to within 5 dB of their pre-exposure levels by 24 hours, there is an obvious continuation of recovery up to 48 hours. Even though there was little more than 5 dB threshold shift at 3 and 8 kHz, recovery at these frequencies was not complete before the 24-hour measurement. There was little, if any, change in threshold for these frequencies the first 8 hours after exposure. Once more, we have evidence that it is not merely the magnitude of the threshold shift that is important for rate of recovery, but also how that threshold shift was produced.

The patterns of development and recovery of TTS for those frequencies at which peak TTS was measured using the octave band of noise centered at 4 kHz, was similar to that observed for noise exposure at the octave 300 to 600 Hz. This relationship is illustrated in the graph in Figure 6. The development and recovery TTS for 4 kHz at the 80 and 85 dB exposure levels from the present experiment is shown together with the development and recovery for 750 Hz measured in the previous experiment. (10) In the earlier experiment, the exposure noise was an octave band 300 to 600 Hz at 90 dB octave band level. The development pattern

for TTS at 750 Hz is almost exactly like that seen at 4000 Hz for the present 80 dB exposure level. The development curve is tri-phasic. The recovery patterns continue to show a decrease in threshold to pre-exposure levels up to 48 hours post-exposure.

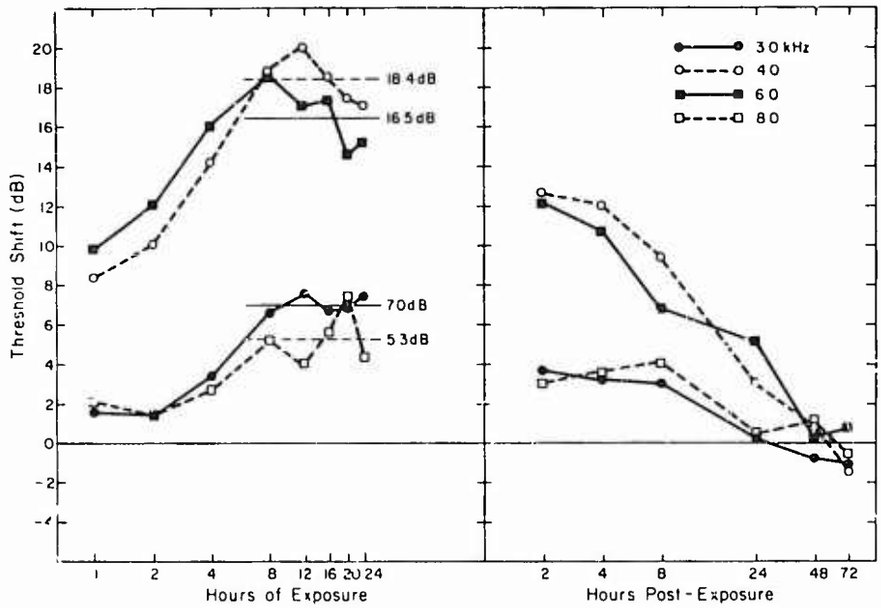

Figure 5

Development of TTS (left graph) and recovery from TTS (right graph) for test frequencies 3,4 6, and 8 kHz from exposure to an octave band of noise centered at 4 kHz at 85 dB octave band level. Data points are the mean for seven subjects.

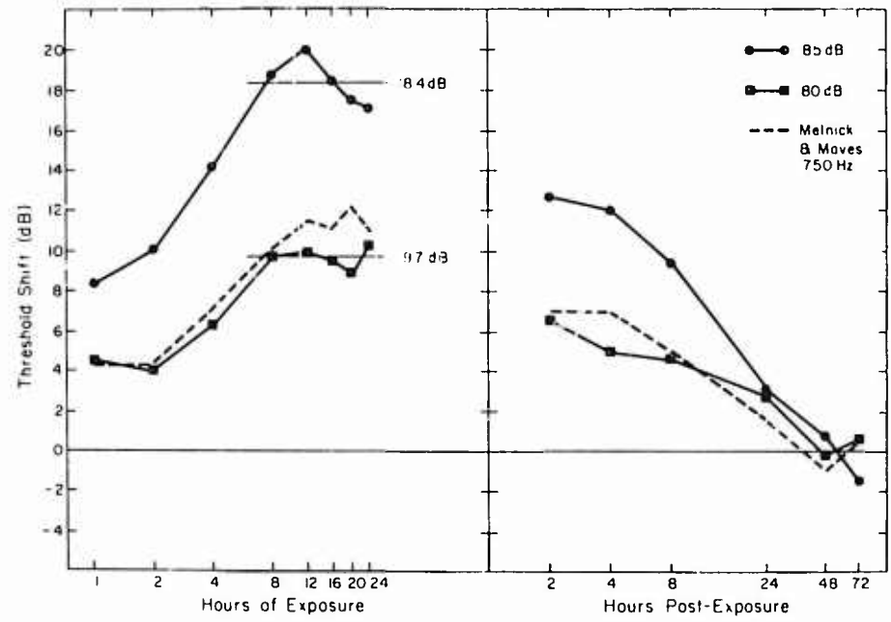

Figure 6

Development of TTS (left) and recovery from TTS (right) for 4 kHz for noise levels 80 and 85 dB. Shown for comparison (as a broken line) are similar temporal patterns obtained for 750 Hz using a 300-600 Hz octave band of noise at 90 dB from an earlier experiment (10) which used ten subjects.

As can be seen from Figures 4 and 5, TTS seems to approach a peak level at somewhere between 8 and 12 hours and then begins to decrease. This decrease in magnitude of threshold shift following a peak level was observed also for frequencies in the earlier experiments with the lower frequency noise. (9,10) The downturn was observed at both the 80 and 85 dB exposure levels for 6 kHz and is observed only at the 85 dB exposure level for the test frequency 4 kHz.

The tri-phasic development curve is more obvious at the lower exposure levels where maximum TTS is not very great. When the magnitude of TTS increases, the early slow component becomes much less obvious. It is conceivable for higher levels of threshold shift that the early component would be entirely obscured.

The data displayed in the previous figures were, of course, group data. There was considerable variation in the growth pattern and the magnitude of threshold shift by individual. Perhaps an indication of the individual variation can be appreciated by graphing the standard deviation of threshold shift as a function of the length of exposure or recovery period. These data are displayed in Figure 7 for the test frequency 4 kHz at both the 80 and 85 dB exposure levels. There was a slight, but systematic increase in the magnitude of variation as the exposure duration increased. Variation in threshold shift among these subjects was related also to exposure level, with the average standard deviation being slightly more for the 85 dB level than for the lowereexposure level. This observation is not surprising, in view of the small amount of threshold shift produced by the 80 dB exposure level in this subject sample. Considering the small magnitude of threshold shift observed in this study, the variable effect of the noise exposure on hearing sensitivity of individual subjects is pronounced.

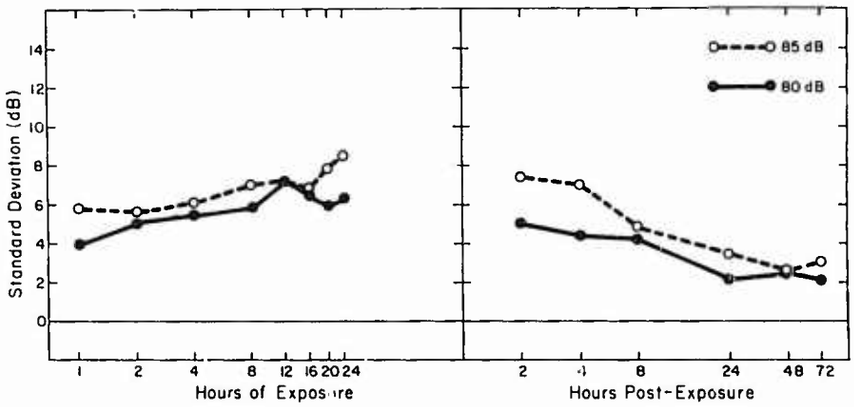

Figure 7
Standard deviation of threshold shifts at 4 kHz measured for seven subjects during and following the exposure period.

During the recovery interval, variation among these subjects decreased as the time following exposure was increased. It approached the level of pre-exposure intra-subject variation, ± 3 dB, by 24 hours of recovery. Individual variation continues to remain a prominent feature of the data from noise-exposure experiments.

Earlier experiments with chinchillas exposed to noise centered at 400 Hz and 4 kHz and earlier experiments with low frequency exposure in humans (2,3,4,7,1,10) indicated a growth of ATS with an increase in exposure level which had a slope of approximately 1.7 at frequencies where peak asymptotic threshold shifts were recorded. For every decibel increase in the octave band level of the noise, there was an increase of about 1.7 dB in the magnitude of ATS. The ATS for 4 and 6 kHz in the present experiment are plotted in Figure 8. The growth pattern for these two frequencies can be fitted very nicely with a straight line having a slope of 1.75, similar to the previously observed growth rate for ATS.

In a report prepared for the Environmental Protection Agency in 1971 by Miller (11), hypothetical growth and recovery curves were plotted for threshold shift measured at 2 minutes after exposure at a test frequency of 4000 Hz when the noise spectrum was centered in that frequency region. These are exactly the conditions of the present experiment, with the exception that TTS for 4 kHz was measured at 6 minutes following noise interruption. Recovery data indicate that this difference should have minimal effect on the amount of threshold shift. Using the hypothetical growth curves that Miller calculated for that report, ATS was plotted as a function of the octave band level for comparison with our own data. This plot is represented as the dotted line in Figure 8. The slope of the hypothetical growth rate of Miller was 1.6, essentially that measured here. However, Miller would have predicted a greater magnitude of threshold shift at 80 and 85 dB octave band levels than we observed with our sample of subjects. His growth curve intersects the 0 dB line at approximately 60 dB, while ours intersects the line at approximately 75 dB. Our data indicate that ATS would be predicted by the equation ATS at 4 kHz = 1.7 (OBL - 75). Quite frankly, the relatively small magnitude of TTS observed in these experiments was surprising. The experiments with chinchillas and previous human experiments with shorter term, higher level exposures would lead one to expect that the magnitude of TTS should have been much greater than was observed here (3,13).

Certainly, there are sufficient number of procedural variables that differ among the various experiments. Most human TTS experiments were accomplished using earphones and not a sound field. This of course would permit greater control over the exposure levels at the ear of the subject than was possible in the present experiment. However, probe tube measurements at the entrance of the ear canal for varying positions assumed by the subjects within our sound field indicated that the magnitude of the noise did not greatly differ from that measured using the microphone in the sound field with the subject's head absent. In fact, because of the diffraction and reflection effects, the magnitude of the sound levels at the entrance of the ear canal of a subject was, in most instances, greater than measured in an empty sound field.

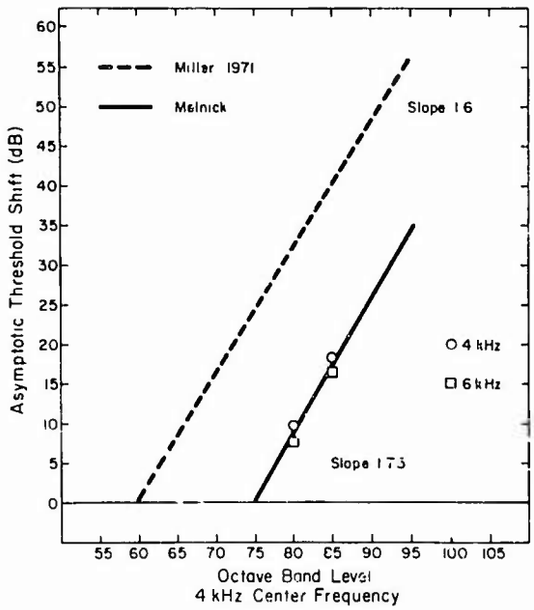

Figure 8
Growth of ATS at 4 and 6 kHz as a function of the octave band level of the exposure noise. Hypothetical values adapted from Miller (11) are indicated by the broken line for comparison.

A more likely reason for the low magnitude of threshold shift in the present experiment is subject sampling bias. Recall that in order to be included in the experiment the subjects had to have thresholds which indicated hearing losses of 10 dB or less at the frequencies above 1000 Hz. In the population available for this experiment, subjects meeting thesecriteria were difficult to find. Most of the people interviewed and tested as possible participants were found to have hearing losses of varying magnitudc which exceeded our acceptance level. It is very possible that by using our criteria, we selected men who are less susceptible to the effects of noise. All of the men who served as subjects were in the 20, 30, and some in the 40-year-old age group. These subjects indicated that they had previous experience with noisy working conditions. However, there did not seem to be any residual effects from these previous noise exposures. If susceptibility is distributed normally within a human population, then perhaps we have sampled from the low-susceptibility tail of the distribution.

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PROTECTIVE EFFECTS IN MEN OF BRAIN CORTEX GANGLIOSIDES ON THE HEARING LOSS INDUCED BY HIGH LEVELS OF NOISE.

Major Dr. G. MANIERO, IAF, MC
Sanitary Group - 1st Aerobrigade - Italy
Prof. G.A. MOLINARI, MC
Department of Otolaryngology
University of Padua - Italy

SUMMARY

It is known that the prolonged exposure to noise of intensity superior to 70-80 decibels determines a temporary raise of the acustic threshold (TTS).

Since Gangliosides are glycolipids which seem to interfere with the transmission of nervous impulse, we attempted, as outlined in the present note, to observe, by means of ganglioside administration, a possible interference on the traumatizing effect of noise on the cochlea.

Following otologic and audiometric examination, 20 healthy subjects were chosen. We calculated, in these subjects, the TTS_2 both in basal conditions and after ganglioside administration.

From our results, the gangliosides, administered in opportune doses and modalities, were capable of preventing in all subjects the physiological rise in the hearing threshold after exposure to noise.

In contrast, the non-treated subjects in the same experimental conditions had either the same TTS_2 or showed a large shift. Therefore, the positive failure in shift, occuring in the treated patients, is most probably due to ganglioside effect.

INTRODUCTION

Exposure to noise of high intensity for a sufficient period of time is known to be capable of determining a temporary, reversible rise of the hearing threshold. Such a rise varies, in a more or less degree, depending on the stimulus parameters and on the cochlea receptor sensibility.

The physiopathological mechanisms responsible for the temporary rise in the hearing threshold are, until now, unknown. Various hypothesis were made to explain such phenomena such as the occurence of fatigue at the level of the nuclei and central cochlea pathways, reduction in activity of the primary afferent neurons, and depletion of the synaptic transmitters between the hair cells and nervous terminations.

Some Authors have demonstrated a decrease of oxygen tension in the endolymph and a modification of the ionic concentration.(1). Other Authors reported a decrease in glycogen, glucose, and enzymes such as succinic dehydrogenase and similar (2-3-4).

The purpose of the present research was to observe if the administration of cerebral cortical gangliosides was capable of reducing in normal subjects the temporary rise in hearing threshold occuring after exposure to high intensity noise. The various data accumulated on gangliosides activity demonstrated that they are capable of returning to normal the nervous conduction altered by pharmacological and traumatic means.

METHOD

The experiment was carried out on 20 volunteer subjects of ages between 19 and 50. The subjects, whose hearing was within normal limits, were completely exempt from otopathies both present or old.

A tonal audiogram was performed on each subject at the frequencies of 0,5 - 1 - 2 - 4 - 8 KH_z.

The deafening acustic stimulus employed was characterized by a noise of low bands centered on 2 KH_z, of intensity equal to 100 db which lasted for an uninterrupted period of 30 minutes. The temporay increase in the hearing threshold (TTS_2) was measured in each subject two minutes after the termination of the noise.

The subjects were divided into two groups. The first group or control group (5 subjects) was submitted to the deafening noise and the TTS_2 was calculated for the two consecutive days without administration of any drug.

The second group (15 subjects) underwent deafening in the morning and successively were administered 2 phials of gangliosides, one 12 hours and a second 1 hour before the repetition of the deafening experiment.

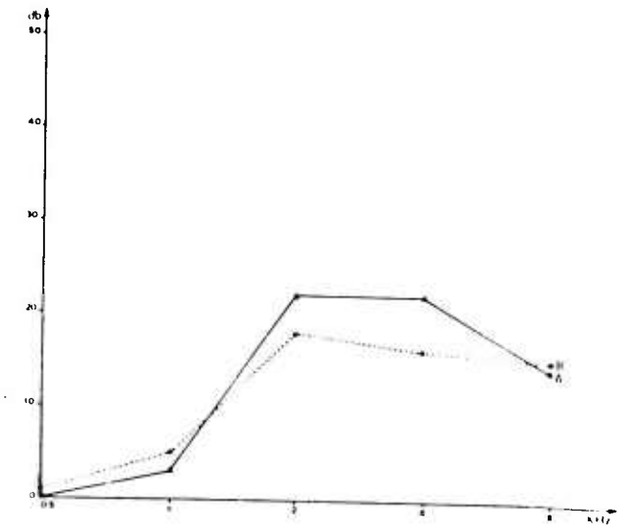
RESULTS

The results obtained are summarized in the following tables and graphs.

KH_z	0.5		1		2		4		8	
YTS_2 After 30 of deafening	1_{99}	2_{99}	1_{99}	2_{99}	1_{99}	2_{99}	1_{99}	2_{99}	1_{99}	2_{99}
N° of subjects										
1 (a 33)	0	0	0	0	20	20	20	10	10	25
2 (a 32)	0	0	0	10	20	20	15	20	10	10
3 (a 31)	0	0	5	5	20	10	30	20	20	30
4 (a 28)	0	0	0	0	30	15	30	15	10	10
5 (a 30)	0	5	10	10	20	25	15	15	10	10
Average	0	1	3	5	22	18	22	16	14	17
Standard Error	0	112	2.23	2.50	2.24	2.85	3.78	2.09	2.74	4.87
T	0.89		0.59		1.10		1.38		0.53	
$P_=$	ns		ns		ns		ns		ns	

Tab n 1

TABLE 1: Table relative to graph. no 1. Values in decibels of the TTS_2 observed in five subjects of ages between 28 and 33 on the 2 days following exposure to noise. (Noise of low bands on $2KH_z$ at 100 db for 30 minutes). (a = age).



GRAPH 1: Temporary rise of the hearing threshold after exposure to noise. Conventionally zero was used to indicate the basal audiogram. The curves of the shift in the hearing threshold after deafening represent the average values expressed in decibels.

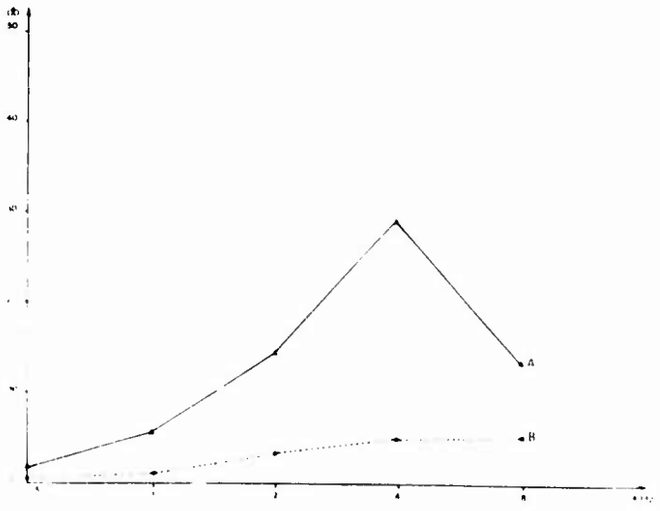
Curve A = TTS_2 observed on the 1st day of deafening.

Curve B = TTS_2 observed on the 2nd day of deafening.

kH_2	0.5		1		2		4		6	
TTS_2 After 30 of deafening	1°dd	2°dd	1°dd	2°dd	1°dd	2°dd	1°dd	2°dd	1°dd	2°dd
N° of subjects										
1 (a 35)	0	0	10	0	10	0	40	0	20	0
2 (a 50)	0	0	10	0	0	0	20	0	10	0
3 (a 21)	0	0	0	0	0	0	20	0	10	0
4 (a 30)	0	0	0	0	20	0	40	20	20	0
5 (a 33)	0	0	0	0	10	0	20	0	0	0
6 (a 40)	0	0	10	0	10	0	20	0	10	0
7 (a 36)	10	0	20	0	20	0	30	10	10	0
8 (a 21)	0	0	0	0	10	0	10	0	30	30
9 (a 26)	0	0	0	0	10	0	30	0	10	0
10 (a 29)	0	0	0	0	10	10	30	20	10	0
11 (a 52)	0	0	0	0	10	0	20	0	0	0
12 (a 33)	0	0	0	0	20	0	30	0	10	0
13 (a 19)	0	0	0	0	20	0	40	0	10	0
14 (a 32)	0	0	0	0	20	0	20	0	10	0
15 (a 48)	0	0	10	0	30	0	30	0	10	10
Average	1.0	0.0	5.0	0	14.0	0.7	28	3	13.3	5.3
Standard Error	1.0	0.0	1.7	0.0	1.8	0.7	2.0	1.2	1.3	1.8
t	0.875		2.50		5.36		8.85		2.37	
P	ns		< 0.02		< 0.001		< 0.001		< 0.01	

Tab n 2

TABLE 2: Table relative to graph no.2. TTS_2 before and after administration of 2 phials of gangliosides in 15 subjects of ages between 19 and 52. (a = age).



GRAPH 2: TTS_2 in the subjects treated with 2 phials of gangliosides. Zero was conventionally used to indicate the basal audiogramm: the curves of shift in the hearing threshold after deafening represent the average values expressed in decibels. Moreover, the maximums and minimums relative to each average value are reported.

Curve A = TTS_2 observed on the 1st day of deafening.

Curve B = TTS_2 observed on the 2nd day of deafening after administration of two phials of gangliosides.

Upon examination of these tables, the following considerations may be drawn. The results of this research confirm that the exposure to noise of low bands on 2 KH_z of intensity of 100 db for a time period of 30 minutes determines a rise in the hearing threshold. Such rise is insignificant at frequencies of 0,5 - 1 KH_z, well-evident at frequencies of 2-4-8 KH_z with a maximum rise occuring at the frequency of 4 KH_z. (see fig. 1,2, and table A and B).

No substantial modifications occur between the TTS_2 observed in the normal subjects on the first day and that observed on the second day. The injection of 2 phials of gangliosides reduced in a well-evident manner the TTS_2 in the second deafening phase.

DISCUSSION

According to the research data of many Authors, the temporary shift of the hearing threshold may be due to a temporary decrease in the capacity of nervous impulse conduction consequent to exposure of intense noise. (Bibliography concerning the modifications in conductance following acustic injury, 5-6-7-8-9-10).

On the other hand, various experiments performed on in vitro models have demonstrated the connection between the transmission of the impulse and the presence of gangliosides in the nervous structures (11-12-13-14).

Such research demonstrated that the functionality of the nervous system, blocked by various means, may be reactivated by the introduction of gangliosides, isolated from the CNS, in the incubation medium.

Experiments done in vivo on models where the stimulus conductance was artificially altered confirmed the pharmacological intervention of gangliosides in the normalization of the conduction phenomenon.

It was also observed that, in man, gangliosides administered intramuscularly were capable of normalizing the various pathological conditions caused by insufficient nervous conduction both at the central and peripheral level.

Our research attempted to demonstrate, in man, the effect of ganglioside administration which is readily observable, in an acute manner, even in an easily analizable experimental pathology.

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* "CRONASSIAL" - FIDIA Pharmaceuticals - Abano Terme Italy
1 phial contains 10 mg of bovine cerebral cortical gangliosides titrated in N-acetylneuroaminic acid (250 mcg/mg).

DISCUSSION

Q. (Nicholson) Could you explain the mode of action of this drug and the manner in which its transmitters affect the physiology of the human body?

A. Its mode of action is not clear. We do know that there are glycolipids which are involved in the neurotransmission but do not know the exact mechanism of action because we do not know which is the neurotransmitter in the cochlea.

Q. Does the administration of this drug impair normal hearing?

A. This is a new drug and we do not have wide experience with it. We have no reason to feel that it impairs normal hearing and, in fact, we had good results following its administration to patients who had impairment of their cochlea prior to administration of the drug.

Q. (Whitcomb) Does the drug have any side effects?

A. No, there are no side effects. For example, blood pressure is not affected. There are some local effects such as pain.

STUDIES OF ASYMPTOTIC TTS

W. Dixon Ward, Ph. D.

Hearing Research Laboratory, University of Minnesota, 2630 University Avenue SE, Minneapolis MN 55414

SUMMARY

Ten young normal-hearing listeners were subjected to a series of exposures to 4000-Hz noise for periods ranging from 2 to 24 hours. The asymptotic TTS (temporary threshold shift) was always reached in 8-12 h, with no suggestion of a sharp increase between 8 and 24 h that has recently been predicted. Little difference could be seen in the rate of recovery from the TTSs produced by 8- and 24-h exposures. It is concluded that exposures longer than 8 h are not unusually hazardous per se; if there is an increased risk of eventual permanent damage from repeated daily exposures longer than 8 h, it probably comes from the fact that as the daily exposure becomes longer than 8 h, the quiet interval before the next exposure--i.e., the recovery period--must perforce become shorter, so that the next day's exposure is begun with the auditory system still in a fatigued state. (Research supported by the National Institute for Occupational Safety and Health, Public Health Service).

INTRODUCTION

When exposed to a steady noise above 70 to 90 dB SPL, depending on its frequency composition, the auditory system develops a temporary threshold shift (TTS) that appears to reflect an underlying exponential adaptation or fatigue process. That is, if one plots TTS in decibels against the logarithm of time, the result is generally a reasonably straight line, at least for exposures up to 8 hours in duration (1,2). Recent results using longer exposures have indicated that this growth does not continue indefinitely, but that after 10 to 12 hours the TTS reaches an asymptote (3,4,5), or at least a plateau that will endure for exposures as long as 15 days.

In principle, then, if one wants to predict the TTS produced by an indefinite exposure to noise at a particular level, one need only actually expose the test subjects for 2 to 4 h, measuring the TTS at various times during the growth process, and then extrapolate this growth to 12 h. The assumption here is that there will be no sudden increase in the rate of growth of TTS after several hours of exposure.

The empirical data all appear to support this generalization as long as the noise levels involved are high enough to produce a measurable TTS_2 (TTS 2 min after cessation of exposure) in an hour or two. Such levels, of course, are the ones that have received the most attention; small TTSs were considered to be of only academic interest until now, not only because of the difficulty of establishing their statistical significance, but also because there was no evidence that such small TTSs could ever lead to permanent loss.

However, in his first study of asymptotic TTS (in himself), Mills (3) reported that the TTS_4 at 750 Hz produced by a 500-Hz octave band of noise at 81.5 dB SPL remained at 3 dB as exposure duration increased from 15 min to 6 h, but then increased to 10.5 dB at 12 h.

That one curve, based on one observer, is to my knowledge the only empirical justification for some "hypothetical curves" published by Miller in his otherwise excellent review of the effects of noise on man (6,7), curves that predict the growth of TTS in a 4000-Hz octave band of noise. The latest version of this graph is shown in Figure 1. Miller predicts that a noise level of only 70 dB SPL will produce a positively-accelerated growth of TTS that will eventuate in a 17.7-dB TTS_4 (three significant figures are of course not justified) after 24 h, and that even 60 dB of noise will lead to 5 dB of TTS. I suspect that a recent decree of the Office of Noise Abatement and Control of the Environmental Protection Agency, in which 70 dBA is proclaimed to be the highest 24-h level that will "protect the public health and welfare with an adequate margin of safety" (8), was based considerably on these extrapolations. In order to test his curves directly, some 24-h exposures to steady 4-kHz (octave band) noise at 75 and 80 dB SPL were undertaken in conjunction with an ongoing study of TTSs produced by intermittent noise.

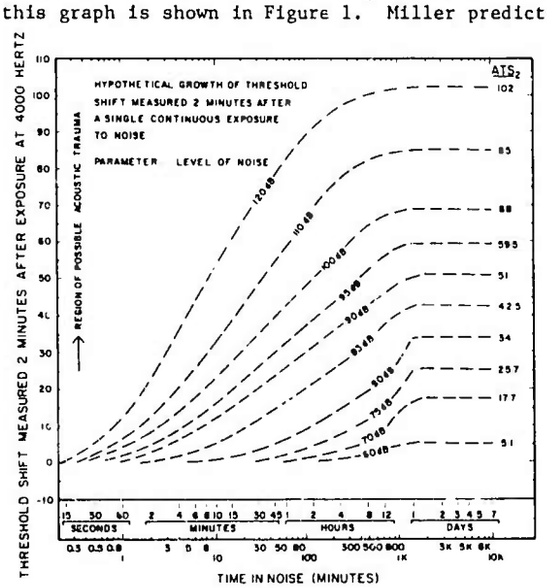

Fig. 1. Miller's hypothetical growth contours; TTS_2 at 5.6 kHz as a function of the level of a 4-kHz octave band fatiguer.

PROCEDURE

Our test subjects are 10 normal-hearing (within 10 dB of ANSI 1969 normal) college students who are paid about $3 per hour for participation; part of their pay is reserved for a lump-sum payment at the end of the school quarter; this bonus encourages completion of a series of test in which each subject serves as his own control. One group of 5 listeners is exposed to noise (usually for 6 h) on Monday and Wednesday, the other on Tuesday and Thursday, generally beginning about noon. Pre-exposure audiometric thresholds are determined daily at half-octave intervals from 0.25 to 11.2 kHz by means of an interrupted-tone fixed frequency Bekesy procedure (250-msec tone pulses with rise- and fall-times of 20 msec, separated by 250 msec of silence; Grason-Stadler recording attenuator running at 4 dB/sec, controlled by the listener; TDH-39 earphones in MX-41/AR cushions). Every 60 min after the beginning of the exposure, each subject leaves the noise for 3 min. After 1, 2, 4 and 6 h of exposure this 3 min is spent being tested at three frequencies (termed "on-frequencies") that usually show the most TTS from that particular noise; beginning 1 min after leaving the noise (which, in the case of intermittent noise, occurs at the end of a noise burst), 20 sec are spent testing each of the three frequencies in the right ear with an XYZ order, then in the left ear in reverse order (ZYX). Thus on average, the TTS at any frequency thus measured is TTS_2. If any individual TTS_2 exceeds 30 dB, the exposure is terminated. During the 3-min break at 5 h, three other frequencies ("off-frequencies") are tested; at 3 h the 3 min is "free". Following the end of the exposure, the on-frequencies are again tested after 15 min of recovery (thus leading to TTS_{17}), and also at 30, 60, 90 and 120 min. The off-frequencies are tested at 45 and 105 min. Finally, 16 h after exposure (i.e. on the morning of the following day), each listener is once again given a complete audiogram. For longer exposures than 6 h, the only modification of the above procudure is to test on-frequencies also after 8, 12, 16 and 24 h of exposure, off-frequencies at 7, 11, 15 and 23 h, and to get a complete audiogram 8 h after the end of the exposure as well as after 16 h.

Noises are generated by a system consisting of a Grason-Stadler white-noise generator, two Allison passive filters, attenuators, and 4 Bogen amplifiers that drive a bank of 64 5-in speakers. The speakers are mounted, 16 to a panel, in a 4-panel "shoji-screen" plywood frame that is situated at one end of our reverberant room. This 15'x18'x12' room, patterned after one at the Acoustics Laboratory in Dusseldorf, Germany, has hard non-parallel surfaces in order to minimize standing waves while retaining a high reverberation time.

During these long exposures, listeners are allowed to sit in comfortable chairs anywhere they like so long as they stay more than a foot away from the walls and at least 3 ft from the loudspeakers (the latter condition is essentially guaranteed by a "movable" concrete pyramid that is placed just in front of the speaker system). Dozing is permitted, as long as the ears remain unprotected. For the 24-h experiments, during which most of the subjects slept from the 16- to the 23-h test, plastic chaise lounges were available. The free 3-min period each hour (free, that is, except when testing was done) was maintained in these long exposures, except of course while the subject was asleep.

The exposures of main interest here were to a nominal 2800-5600-Hz noise. However, because of the rapid drop in response of the speaker system above 4000 Hz, the actual 3-dB-down frequencies of the noise were 2800 and 4600 Hz. Levels were measured and monitored with a 1/2-inch condenser microphone (Bruel and Kjaer Sound Level Meter Type 2203); the variability of the noise field from place to place in the listening area was ± 1 dB. A continuous record of the exposure was made using an Altec 150BR microphone feeding a Bruel and Kjaer Level Recorder.

One group of 5 listeners was exposed to this noise at 75 dB SPL, the other at 80 dB SPL. Temporary threshold shifts were calculated relative to a mean pre-exposure threshold that was determined by averaging the values obtained over a period of 2 to 3 months (i.e., based on at least 10 different pre-exposure audiograms).

RESULTS

The filled circles in Fig. 2 indicate the results of the exposure at 75 dB SPL, expressed in terms of the average of the TTS in right and left ears at 4 and 5.6 kHz, the two frequencies most affected (and equally affected) by this noise. It is clear that the drastic increase of TTS predicted by Miller (long-dashed lines) after 8 h does not occur. Indeed, in this case, asymptotic TTS is essentially achieved in the first hour of exposure.

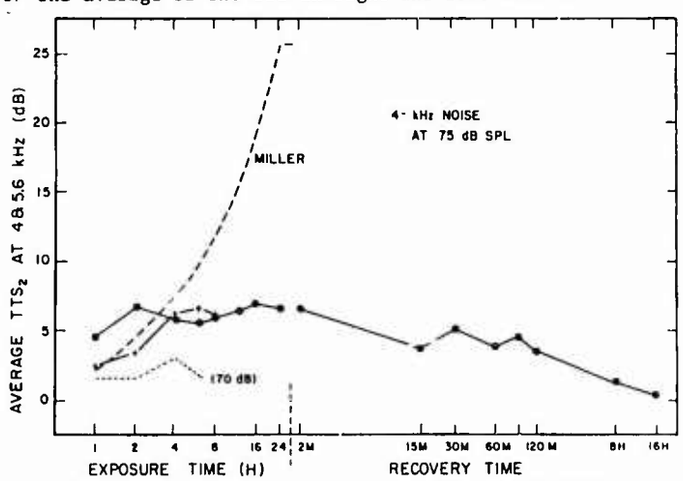

Fig. 2. Growth of TTS_2 with time for 70- and 75-dB levels of a 4-kHz octave band fatiguer.

Also plotted in Fig. 2 is another exposure to this 75-dB-SPL noise. The crosses show the TTS_2 (again, averaged over right and left ears and over 4 and 5.6 kHz) produced by an 8-h exposure in a different group of 10 subjects 2 years earlier (the 1972-3 listening crew). Finally, the dotted curve at the bottom shows the TTS_2 produced by a 6-h exposure at 70 dB SPL to yet another group of 10 different listeners (the 1973-4 listeners).

The process of recovery is shown on the right.

The lower sets of curves in Fig. 3 represent the results of exposure to 80 dB. Again, the filled circles indicate TTS from the present 24-h exposure, the long-dashed line Miller's "prediction", and the crosses two different 8-h exposures (separated by 7 months) of the 1972-3 group. Also shown are two 6-h exposures; open circles for the present group (1974-5), triangles for the 1973-4 group. The results speak for themselves: the asymptotic TTS_2 produced by 4-kHz noise increases from about 6 dB for 75 dB SPL to around 12 dB for 80 dB SPL, with no sign of any increase after 8 h of exposure. Furthermore, recovery is complete after 16 h of quiet.

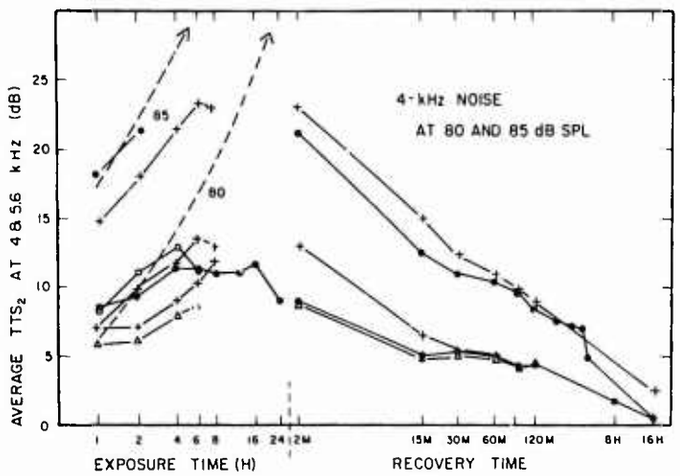

Fig. 3. Growth of TTS_2 with time for 80- and 85-dB levels of a 4-kHz octave band fatiguer.

What happens at 85 dB must be left partly to conjecture. In the only attempt to test continuous exposure at this level, one of the subjects in the 1972-3 group developed a TTS_2 after 6 h that exceeded the 30-dB ceiling placed on TTS_2, so the exposure was terminated after 7 h. However, extrapolation implies that the asymptotic average value should be about 25 dB. A 2-h exposure of five of the 1972-3 listeners was also run a few months later. These results, together with Miller's prediction, are shown in the top left part of Fig. 3. Recovery curves on the right indicate that the 7-h exposure produced a TTS that lasted more than 16 h; this was due to three ears that still showed 12 to 18 dB of TTS at this (recovery was complete at the test 24 h later.)

It will be noted that there is, in many of the curves, a drop in the TTS_2 at the termination of exposure, whether this is at the end of 6, 8, 12 or 24 h. In the 24-h exposures this may be due partly to a decrease in the effective level of the noise during the sleep period from 16 to 23 h (we could have tried to make them sleep only on their backs so that both ears would be continually uncovered, but this was thought to be neither reasonable nor realistic). However, for other durations of exposure, this decrease in TTS_2 at the end of exposure is what one might call a "thank God that's over!" artifact. Apparently these subjects listen a bit more carefully when they know that they do not have to return to the noise. Although such an effect was not apparent in some 8-h exposures done in Germany (2), it can be seen in other American long-term exposures (4,5). Differences in motivation are no doubt involved here.

DISCUSSION

Figure 4 represents a revised prediction, based on the present data, of growth of TTS_2 after exposure to octave-band noise centered in the 3- to 4-kHz region, one without the bizarre acceleration of effect between 8 and 24 h postulated by Miller. No attempt is made to predict exposures that would lead to average TTS_2s of more than 40 dB, since they would be almost universally thought to be hazardous. A comparison of Fig. 4 with Fig. 1 indicates that, for levels of 85 dB or less, there is essentially a 10-dB error in Miller's estimates of what SPL will just produce a specified asymptotic TTS_2. This is an error of not inconsiderable magnitude, considering that, as mentioned earlier, Miller's curves have been interpreted as providing support for the proposition that 24-h exposures to 70-dBA noise--and hence, applying the so-called "equal energy" principle, 8-h exposures to 75-dBA noise--can lead to permanent posses (8).

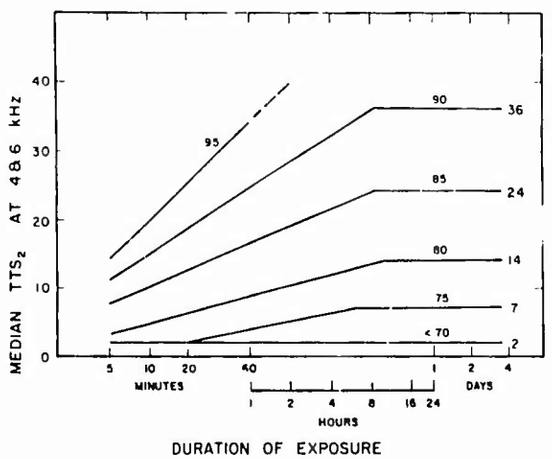

Fig. 4. Revised idealized growth curves for TTS_2 at 5.6 kHz following exposure to 4-kHz octave-band noise at various levels.

Let us therefore first discuss the situation in which exposure is 8 h of continuous noise followed by 16 h of quiet. The present data indicate that even if there were such an industrial noise as one with all its energy in the 4-kHz region, exposure to 75 dBA (ca. 74 dB SPL) will lead only to an average TTS_2 of less than 15 dB, with full recovery after 16 h of quiet. If we accept as a criterion for a "safe" exposure the production of no more than 30 dB of TTS_2, and if we can assume a standard deviation no higher than 6 dB among individuals when the mean TTS is 12 dB (9), then about one person in 1000 could be considered "at risk" in an 80-dB-SPL noise. As a matter of fact, the highest asymptotic TTS_2 (the average of TTS_2s after 8, 12 and 16 h of exposure) at any frequency in any ear in this group of 5 listeners was 19 dB. Furthermore, the foregoing is for the worst condition in terms of spectrum; more commonly an industrial noise with a 4-kHz octave-band level of 80 dB SPL will have an overall A-weighted level of 86 or 87 dBA.

The implication of the foregoing line of argument is that habitual 8-h exposures to an industrial noise whose A-weighted level is 86 dBA should produce no permanent hearing loss, if it is true either that disappearance of TTS indicates that no residual damage exists or that daily production of up to 30 dB of TTS_2 is never hazardous, the latter provided that no additional noise exposure outside the work situation occurs to reduce the 16 h of normal recovery time.

But now let us consider the recovery process at greater length. Figure 4 shows essentially that the course of recovery from 24-h exposure to steady noise is not significantly (importantly) different from a 6- or 8-h exposure: that is, a 16-h period is sufficient for full recovery, provided that the initial TTS_2 does not exceed 25 or 30 dB, a conclusion already reached for 2-h exposures some time ago (10). The trouble with industrial exposures longer than 8 h, therefore, is apparently not so much that the overtime produces any measurable increase in TTS, but rather the unavoidable fact that if one works a 12-h shift, then only 12 h instead of 16 are available for the ear to recover before the worker enters the noise again.

The extension of the noise exposure beyond 8 h need not, of course, be because of overtime work. Commuting by motorcycle, subway or noisy car, mowing one's lawn, listening to music, using powered hand tools, etc., are all activities that develop levels that can interfere with the recovery process and reduce the time available for recovery, even though the additional exposure itself might be innocuous in isolation. Such noise exposures are commonly termed "sociacusic".

The role of recovery time is minimized in the "equal-energy" theory that is being assiduously promoted in the USA and abroad by those who so earnestly wish it were true, because it would make prediction and measurement of hazard very simple. The most extreme form of this hypothesis, as advocated by Robinson (11), simply integrates A-weighted energy over all time, so that presumably one 8-h exposure at 100 dBA poses the same hazard as 100 days of 8-h exposure to 80 dBA or 40 years (10,000 working days) of habitual exposure at 60 dBA. There is, in this scheme, no room for the concept of a "critical level" of stimulation for a given ear, a level below which no hazard exists no matter how long the exposure. No account is taken of the recuperative powers of the normal auditory system, which seems as silly to me as assuming that the eye integrates visual energy over all time--if this were the case, we would all be blind by adolescence.

The position taken by ONAC is the "Levels" Document (8) is not quite so extreme, probably limiting equality of exposures to an integration over a single day*, which allows one to ignore 8-h exposures to levels below a critical value. Unfortunately, however, this critical value has been set at 75 dBA instead of the 86 or 87 dBA implied by the present data, due in part to the use of a safety factor in order to try to protect "everyone", and in part to acceptance of dubious evidence that 8 h of a 80-dBA noise can produce a measurable hearing loss (12). I have argued elsewhere (13,14) that the extant data imply that a level of 80 dBA is completely innocuous, and that habitual exposure, 8 h/day, to 85 dBA will produce only a permanent loss of 10 dB at 3000, 4000 and 6000 Hz after many years, on average. When one considers that the relevant audiometric data were gathered on workers most of whom, if not all, often "extended" their daily exposure through sociacusic influences, it is not unreasonable to suggest that even these minor (and insignificant in a practical sense) hearing losses are not due to either the industrial or the sociacusic exposures alone, but to their combination, so that 90 dBA may well be the point at which an 8-hr industrial exposure alone would become just barely hazardous.

Let me be more explicit. At the risk of being shot down in flames for my extrapolations by actual data, just as the present evidence has done for Miller's, I am willing to predict that if 1000 normal-hearing workers were exposed for 10 years to a 90-dBA typical industrial noise (i.e. one in which the SPL of the 4000-Hz octave band did not exceed about 83 dB) for 8 h daily from Monday through Friday, but with no sociacusic noise exposure above and beyond that involved in the process of ordinary conversation, the average hearing levels at even 4000 Hz would be no more than 10 dB higher than in a control group, and that no more than one individual of the 1000 would have a loss judged to be handicapping.

On the other hand, I would certainly not predict that the same would be true of a group exposed for 16 h each workday to 87-dBA noise (i.e. the same daily total energy). In going from 8-h to longer daily exposures, even the total-energy concept might be under-conservative because of the shortening of recovery time. Certainly I would not be so brash as to argue that 85 dBA could be tolerated without risk 24 h/day for an indefinite period, although the total-energy principle would imply this (provided that 90 dBA for 8 h is safe, of course), Although most of the results of prolonged exposures of chinchillas imply that the recovery process, once quiet is available, is little affected by how long the asymptotic level was held (15), some data indicate that this may not be true if the asymptotic TTS is maintained for three months (16).

In man, Mills (3) showed a delayed recovery after only 2 days in low-frequency noise, as did the prisoner-subjects exposed by Melnick and Maves (5). Finally, in a study reported only very sketchily, Yuganov et al. (17) indicated that when men were kept for up to 30 days in "a high-frequency noise" of 75 dB, the TTSs at high frequencies after 24 h were 10 to 20 dB, but after 10 days were 20 to 25 dB, and after 30 days were 25 to 30 dB, and required 50 h for full recovery. No effects, however, were produced by similar exposures at 65 dB. It may well be that 70 dBA is the correct limit for an indefinitely long exposure, just as ONAC avers, even though their 8-h proposed limit of 75 dBA is off by at least 10 dBA, if not 15.

It is unlikely that these predictions will ever be tested, not only for obvious practical reasons, but also because it is no longer permitted to expose people to 90 dBA for 8 h at work. It is hoped, however, that analogous experiments can at least be performed on experimental animals.

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* I say "probably" here, because the Levels Document has some ambiguous references to an "annual average L_{eq}", which would imply that for some unspecified purposes one might measure the total A-weighted energy entering the ear over an entire year.

CONCLUSION

In summary, evidence has been adduced showing that asymptotic TTS is reached after 8-12 h of exposure to continuous noise, so that, in an industrial situation, the hazard associated with longer exposures is probably due to the shortening of the available recovery time before the next exposure. A corollary to this conclusion is that the noises of everyday life (sociacusic noises) contribute more than a negligible amount to the growth of permanent hearing losses in workers whose industrial exposures are just at the borderline of hazard. If industrial 8-h noise exposures were held to 85 dBA or below, and workers were prevented from listening to loud sounds outside the work situation, then it appears highly likely that no measurable permanent losses would appear. Even at 90 dBA, the increase in permanent loss might well be unimportant.

This analysis, of course, does not settle the issue of what exposure levels industry "should" be required to adopt. Even though it is not reasonable to blame industry for all the hearing losses that occur in 90-dBA noise, it is just as unreasonable to allow the industrial exposure to be so great that any additional exposure to high levels in ordinary life would tip the scales to permanent loss. How much allowance for sociacusic noises--which are, more often than not, desirable in the eyes of the worker--should be made in establishing maximum industrial exposures?

Industry predictably takes the view that the maximum permitted 8-h exposure should be one that will just not be hazardous in the complete absence of sociacusic influences; the more fanatic representatives of the so-called "public interest" groups (including the framers of the Noise Control Act of 1972) will no doubt continue to try to make it 10 or 15 dB lower, so that any losses that do occur could unequivocally be ascribed to socacusis. A reasonable compromise, it seems to me, would be available if we only had the ability to determine the level that, if maintained for 16 hours, 5 days per week, would just produce the criterion hearing loss. The permitted industrial 8-hour exposure would be set at this level. Under this system, employees would use up only 50% of their noise dose limit at work; limiting their sociacusic exposure to the remaining 50% would be their responsibility.

Unfortunately, we do not have any significant data on men who work at two noisy full-time jobs, so we must use experiments with laboratory animals to infer what this limiting 16-h daily exposure might be. It appears, however, that it would be well worth the effort.

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DISCUSSION

Q. (Lim) I recall Dr. Ward brought up a question concerning one of my figures wherein animals who develop temporary threshold shifts but not permanent threshold shifts nonetheless had evidence of sensory cell degeneration. Behavioral studies with humans and animals fail to show evidence that the cumulative effect of temporary threshold shift leads to permanent threshold shift. I didn't intend to show this figure to emphasize sensory cell degeneration by acoustic stimulation and furthermore I think it is erroneous to try to interpret animal data with human data because animals function at one level and humans at another and we cannot directly interpret one from the other. Secondly, there are some studies on humans performed in other laboratories where it is shown that even a very young individual who does not have any history of acoustic overexposure still shows sensory cell degeneration. This brings us close to a question. Does the human ear start to deteriorate at the time of birth, and continue? Some people think it does. Then a second question evolves. That is, we are given a certain number of sensory cells and we keep losing these cells, but they have never been replaced. And the total number of sensory cells we have in the hearing of one person maybe are not all needed for normal hearing and there might be enough sensory cells that one is allowed to lose some without developing any hearing loss. So the question that we still have to answer is: How can sensory cell losses be shown behaviorally? Also, do we really have adequate behavioral tests, or do the physiological tests that we have today really measure the spectrum of sensory cell function? I don't know that this is an answerable question.

A. (Ward) Well, actually I am not sure that it's an answerable question either. We would like to find the answers to these questions of how much hair cell loss leads to an audiometric change. There is no question that a few missing hair cells will not lead to behavioral deficit. This has been established in many laboratories. Perhaps, we are being unduly risky in waiting until we get a permanent threshold shift before we say that hearing damage has been done. This really isn't the question that I hope we address in our experiments. What we want to do is to determine the exposure that will produce a measurable hair cell loss. The only trouble is that this isn't the correct criterion either because, as Dr. Lim I am sure knows, there are experiments in which moderately severe (40 - 50 dB) hearing losses have been developed at 4,000 Hz in monkeys and in cats; and yet when they inspect the cochleas, the hair cells are apparently intact. So it is even worse than Dr. Lim makes it out because the hair cells may be there but still not functioning properly. I am not sure there is any solution. If we can't find a criterion for the beginning of damage that we can agree on then of course we can't determine what the physical correlates are of the noise exposure that produces that criterion damage. I don't mean to imply that I don't believe in the microtrauma theory of hearing at all. I just always want to emphasize that the auditory mechanism is designed to accommodate to a certain extent and, therefore, it is not necessarily true that merely because the noise exposure produces a 5 dB temporary threshold shift one should expect to find a 5 dB (in a manner of speaking) permanent threshold shift after many years of exposure. This point was established on the basis of measurements at 3 industries. One was a can manufacturing operation, one was a glass manufacturing plant that involved noise of compressed air, and the third was a sewage disposal plant. On the basis of temporary and permanent threshold shifts at 4,000 Hz which amounted to 20 dB or greater, there was a congruence in this case in these three industries. The approximate permanent threshold shift after ten years or more of exposure was approximately equal to the temporary threshold shift produced by a single day's exposure to young normal ears. Now this finding has been extrapolated to conclude that if any temporary threshold shift is produced then one can expect permanent threshold shift eventually. I think that this is an unjustified extrapolation, and this is the point I was trying to make, that when you are trying to determine the lowest possible safe level, it becomes very difficult because, as I say, I cannot bring myself to believe that even a 10 dB temporary threshold shift, repeated day after day, will necessarily lead to any permanent damage. Now that's different, though, from saying that a noise level that on the average produces only a 10 dB temporary threshold shift will never lead to permanent hearing loss in anyone. We have got to consider individual differences. In a situation where the average temporary threshold shift is 10 dB, there will be the occasional person who gets a 20 or even a 30 dB temporary threshold shift. If this person, in addition, happens to be one who likes to run a chain saw, or a power mower, or has power tools at home, then he is going to get a permanent hearing loss; so the distribution is, I guess, the problem here. It would be nice if we could establish that if the temporary threshold shift in the individual ear did not exceed "X" dB, then this would not lead to permanent changes that would be measurable either behaviorally or histologically.

Q. (Henderson) I have a question for both Drs. Ward and Melnick. Both of your sets of data had a great deal of variability in them and this is different from the chinchilla data. Would your data look different if you plotted them in terms of dB sound pressure level rather than decibels of threshold shift? (ref. C-2 Melnick)

A. (Ward) I've plotted my data both of these ways and it doesn't make any difference in terms of the amount of variability shown. In other words, it is not the case, that among those people who are regarded as normal, that the people with the highest threshold get the least shift so that they all come together after exposure.

A. (Melnick) I have found the very same thing.

Q. (Money) It seems that most people who have a social hearing loss are in their sixties and seventies. This gives rise to two questions which perhaps any of the speakers can answer. Is it possible that noise exposure early in life leads to hearing loss later? Are old people more susceptible to hearing loss from noise than young people?

A. (Melnick) Presbycusis has received a great deal of attention lately. Several factors are involved. You have probably hit upon one of them. Some people feel, and quite rightly, that presbycusis is an accumulation of noise exposures throughout a lifetime and that it is almost impossible to separate the long term repeated noise exposure of these people from their aging process alone. There must be an aging process which occurs just as one's skin becomes less resilient, and one can't run 100 yards in 10 seconds when one is older. The same thing happens with the auditory system. It is probably related to such things as the aging of the vascular system. There is a general gradual deterioration from many causes. Sociocusis is the term Dr. Ward mentioned today regarding non-occupational noise exposure. It is important for us to define what we mean. If we mean that hearing loss occurs only from noise during employment, we are wrong. The hunter, the skeet shooter, the military exposure which everyone in this room has certainly had, and the aging process all add together. At age 60 or 70 it is almost impossible to say which one caused hearing loss. In answer to your second question, apparently older people are less resilient to the effects of noise exposure.

A. (Ward) I am not sure that there is any evidence for that last statement that older people are less resilient to the effects of noise exposure. It is quite often assumed that very young ears are more susceptible, or it is sometimes assumed that old people are resistant, or sometimes that old people are more susceptible. How do you do the experiment to prove it one way or the other? That is the problem. Some years ago John Dougherty had a group of old veterans who still had normal hearing. He wanted to do an experiment on them to compare them to younger people. He divided his population into normal old people and normal young people. He planned to see if the two groups got different amounts of temporary threshold shift when exposed to the same noise. The trouble was, how could the experiment possibly come out to prove once and for all whether or not the old or the younger are more susceptible to noise? If the older group showed less effects you could either say that they were less susceptible or that they had tougher ears and they proved that because they had gotten that old without having a hearing loss. This is the problem that one has if one tries to decide how one could go about showing whether an old ear is more or less resistant to further damage. Now to consider Dr. Money's first question. As far as anyone knows, there are no latent effects I think that's what he was asking. That something that happens today, if it has no effect now, will it have an effect later? There is no good evidence that latent effects exist. We have been looking for them for a long time. It appears that once a person is taken out of a noise environment there is no further progression of the damage that has already occurred.

ASYMPTOTIC BEHAVIOR OF TEMPORARY THRESHOLD SHIFT DURING EXPOSURE TO LONG DURATION NOISES

D. L. Johnson, Lt Col, C. W. Nixon, Ph.D. and M. R. Stephenson, 1 Lt
Aerospace Medical Research Laboratory
Wright-Patterson AFB, Ohio 45433

Summary

Exposure to a constant noise level for more than 16 hrs has been shown by many investigators to cause a Temporary Threshold Shift (TTS) in hearing that remains constant. This behavior, which is independent of exposure duration, is called Asymptotic TTS. Data is given which show that although TTS may remain constant, the recovery of hearing back to normalcy does depend on the duration of the exposure. Significant differences in recovery between a 24 hr exposure and a 8 hr exposure were observed. It is believed that for hearing conservation purposes, the time personnel should be allowed to recover from long duration noise exposures in quiet depends on the exposure duration. Suggested guidelines for assuring recovery of Asymptotic TTS are given and the current research program aimed at improving these guidelines is discussed.

Foreword

Currently, personnel of the Aerospace Medical Research Laboratory are attempting to clarify some of the effects of long duration noise exposures on hearing. This research is being performed both to satisfy Air Force requirements, and to support the needs of the Environmental Protection Agency. Such research is directly applicable to many Air Force long duration missions as well as to environmental problems associated with the civilian populace as a whole.

This paper will address three main items. First, some of the essential elements that are known about long duration exposures will be summarized. Second, our current research program that is aimed at some critical factors that are not understood about long duration exposures will be discussed. Included will be some yet unpublished results that bear directly on one of these problems. Finally, a proposal is offered as to how long duration exposures should be treated in the hearing conservation program at the operational level.

Long Duration Exposures, the Asymptotic Behavior of TTS

In the past few years, researchers have begun to focus on long duration noise exposure studies in which both animal and adult humans have been used as subjects. The human experiments that use Temporary Threshold Shift (TTS) in hearing induced by noise as a measure of the damage causing potentialities of the noise have shown one very important fact. This fact is that during the first 8 hrs of continuous noise exposure at a constant level, Noise Induced TTS (NITTS) increases up to a point with increasing exposure duration. At some time, between 8 to 16 hrs of the exposure, NITTS for a constant exposure level does not increase further. This aspect of TTS from long duration exposures has been commonly referred to as Asymptotic TTS. This behavior pattern is seen in Fig 1 as the plateaus in the growth curves between 8 and 16 hrs.

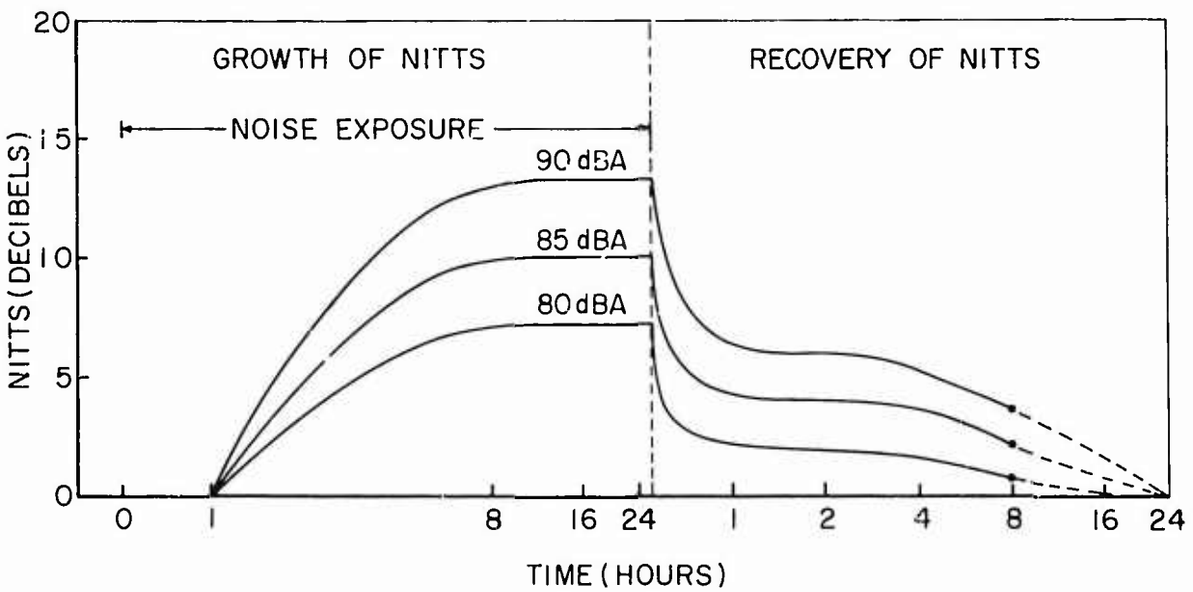

Figure 1. Growth and recovery of Noise Induced Temporary Threshold Shift from 1/3 octave band of noise centered on 1000 Hz. NITTS is an average of the levels at 1000 Hz, 1500 Hz and 2000 Hz test frequencies measured at times marked on abscissa (from ref 11).

Asymptotic TTS behavior has been observed, in both humans and animals, in all the many studies reported (1-11). Such behavior leads to the speculation that there may be a natural protective action in the auditory system that limits the effect that any one exposure level can produce. Carder and Miller proposed that "For a wide range of conditions, Asymptotic TTS probably represents an equilibrium between fatiguing and restorative processes (4)." If this is true, then the implications are obvious. Exposures longer than 24 hrs would not be any more harmful than exposures of 8 or 16 hrs. Thus the criteria for an 80 hr mission in a noisy aircraft would be the same as the criteria for an 8 hr one. This proposition requires us to look more closely at the hypothesis that Asymptotic TTS behavior is a protective action.

There are two aspects of this behavior which warrant further discussion. First, behavioral responses and histological studies can be made of the ears of animals exposed to long duration noises and second, the recovery patterns following such exposures can be studied. Histological studies of animals have shown that after long duration exposures physiological injury sometimes occurs even though the behavorial auditory thresholds are normal. This damage includes hair cell loss, decrease in cochlear microphonics, and decrease in whole nerve action potentials (13, 14). One of the problems with histological studies is that there is not yet enough data to obtain good correlation between graded exposure level and physiological changes. Then to complicate matters, the significance of small physiological changes is not always clear. Perhaps behavioral auditory threshold may not be the perfect measure of auditory function, but as yet a more meaningful measure has not been defined. The physiological changes do indicate the caution and concern with which long duration exposures should be viewed.

The other measure with which the effects of long duration exposure can be investigated is the recovery pattern. Based on animal studies, the recovery pattern is clearly dependent on the duration of the exposure when the Asymptotic TTS is above 55 dB (8). In fact, some animals showing this amount of loss received a permanent threshold shift as recovery was never complete. On the other hand, there was no difference in recovery patterns between exposures of 2 days, 7 days or 21 days, when Asymptotic TTS level was a more reasonable 30 dB (4). Somewhere between ATS's of 55 dB and of 30 dB is a point at which duration of exposure becomes important for that experimental animal. It is reasonably well known that one time noise exposures which cause large values of TTS are dangerous. Therefore, it is not at all surprising to see

permanent injury for some ears that had ATS above 40 dB. The more important part of this experiment was that for moderate amounts of TTS, which we will consider to be 30 dB or less, recovery was independent of exposure duration. The biggest reservation of this study was that it did use animals (specifically chinchillas) and the question that invariably arises is how well does this apply to humans, especially in light of the decrepancies between hair cell damage and behavioral auditory thresholds. Therefore, the verification of the behavioral response phase of this result on humans become the top priority of the research in progress at our laboratory Some results of this program will be reported in the next section.

Current Research Program at AMRL

Current research at AMRL is directed to answer three main questions about ATS, (1) is duration of moderate exposures important once the asymptotic value is reached, (2) what is the threshold of any Asymptotic TTS and (3) what does interruptions of the noise exposure do with respect to Asymptotic TTS behavior?

The first question has been answered by the studies recently completed. Unfortunately, the answer to this question is yes. Using the procedure outlined in Ref 1, a significant difference was found between the recovery patterns following a 24 hr exposure of 11 college males and a 48 hr exposure of the same 11 subjects. Figures 2 and 3 best illustrate these differences for the 4000 Hz audiometric frequency. Table 1, summarizes the differences between corresponding 24 and 48 hr data for each test period as analyzed with the t-test for related measures.

		TTS_2			$TTS_{4.5}$			$TTS_2 + TTS_{4.5}$	
		$\bar{D}$	T		$\bar{D}$	T		T	
Noise Exposure	1	-3.1	-2.3	<.05	.4	.4	NS	-1.6	NS
	2	-6.2	-2.8	<.05	-1.3	-.8	NS	-2.6	<.05
	4	-3.6	-1.4	NS	-1.2	-.5	NS	-1.4	NS
	8	1.3	.7	NS	1.0	+.6	NS	1.0	NS
	16	2.4	1	NS	-0.7	-.3	NS	0.5	NS
	24/48	2.9	1.4	NS	1.2	.5	NS	1.3	NS
Recovery	1	2.1	.9	NS	2.2	1.4	NS	1.6	NS
	2	2.8	1.7	NS	3.9	2.4	<.05	3.0	<.01
	4	3.5	2.5	<.05	4.6	4.4	<.01	4.7	<.01
	7	3.1	2.4	<.05	3.7	3.2	<.05	4.0	<.01
	8	1.6	1.6	NS	1.6	1.3	NS	2.0	NS
	24	2.5	2.3	<.05	2.2	1.3	NS	2.4	<.05

Table 1. Summary of Results Using T-Test For 24 Hr and 48 Hr Data

D = TTS of 48 hr study - TTS of 24 hr study

$\bar{D} = \Sigma D/11$

Note that TTS was measured both 2 minutes and 4 1/2 minutes after the end of the exposure. It appears that either measurement provides an adequate description of the TTS, although TTS 4.5 was found to be less variable. The implication of this difference is that for exposures in the range of 85 dBA, longer recovery times are indicated for longer exposure durations. It can be surmised from Figures 2 and 3, that the TTS from the 48 hr exposure is approximately the same after 24 hrs of recovery as the TTS from the 24 hr exposure after 8-16 hrs of recovery. Thus the 48 hr exposure is causing the TTS to take approximately twice as long to recover as the 24 hr exposure.

Observation of the individual data of the 24-48 hr experiment, reveals considerable variability among subjects. The actual ATS values for 12 subjects varied for the 85 dBA exposure from no TTS to as much as a TTS_2 of 30 dB. Figure 4 is the average of the 4 subjects showing the greatest TTS at 4000 Hz. From this figure we observe that there is not much difference between the behavior of the growth and recovery patterns from TTS for these most susceptible subjects and the average growth and recovery patterns of the entire 11 subjects shown in Figures 2 and 3. The problem of the more susceptible individual appears to be one of larger magnitude of TTS, and not that such a person fails to recover from such TTS.

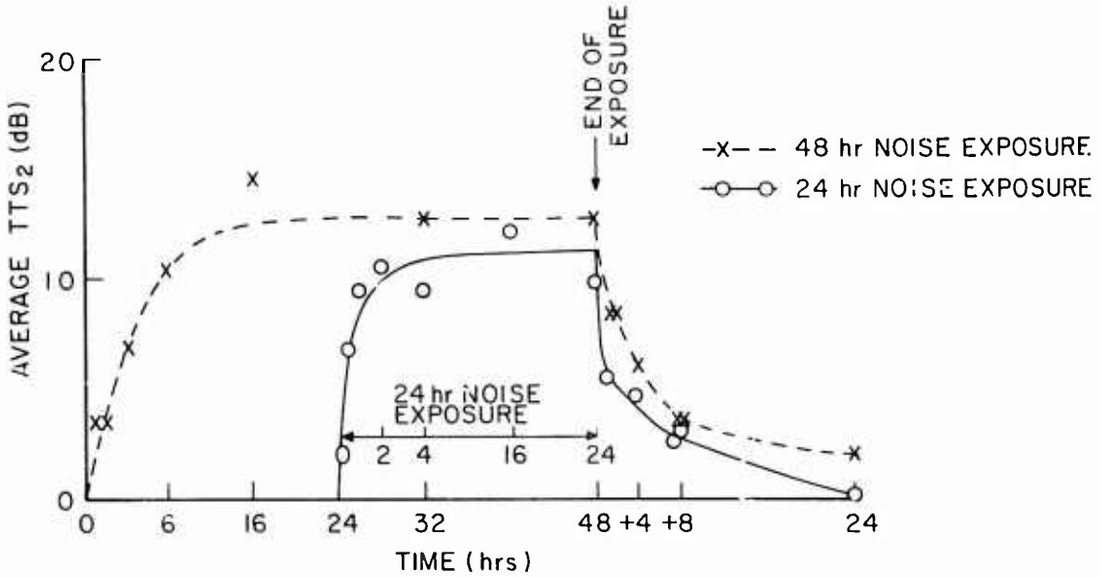

Figure 2. TTS_2 at 4000 Hz from 24 hr and 48 hr exposures of pink noise at 85 dBA (average of 11 subjects).

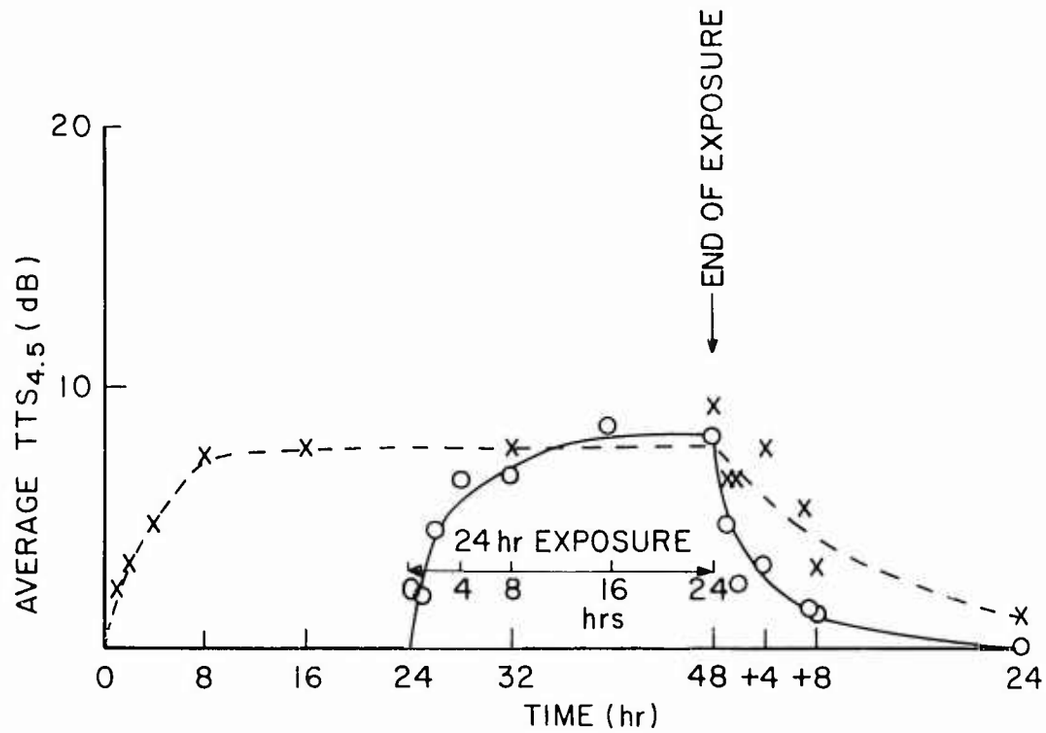

Figure 3. $TTS_{4.5}$ at 4000 Hz from 24 hr and 48 hr exposures of pink noise at 85 dBA (average of 11 subjects).

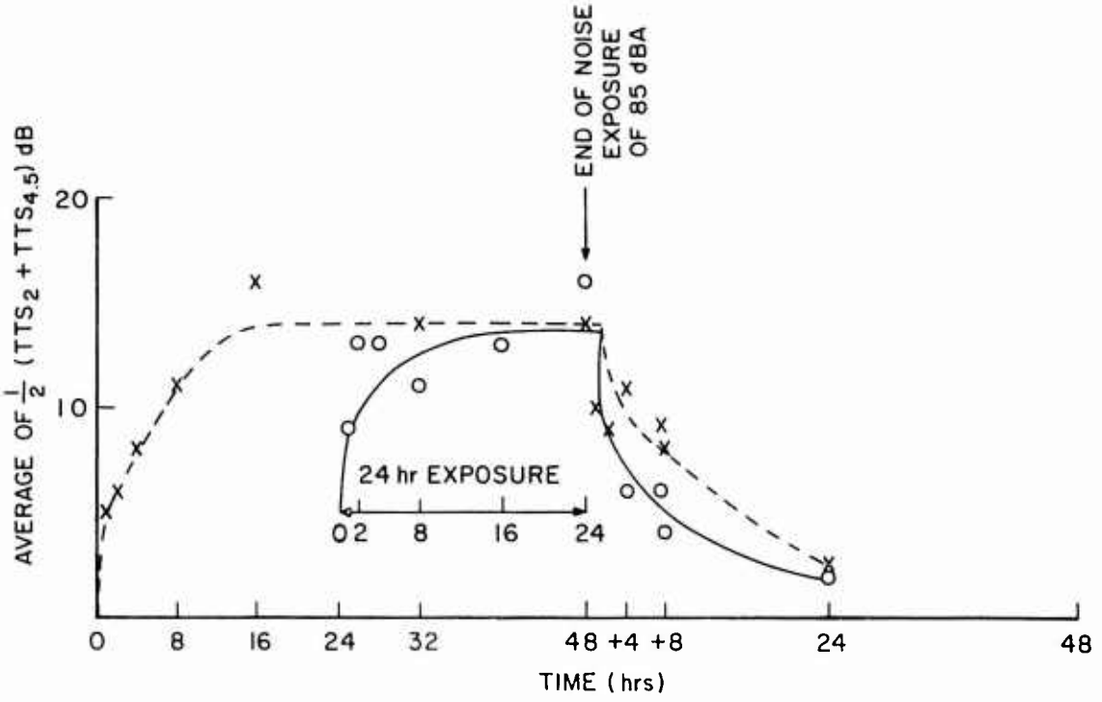

Figure 4. 1/2 (TTS_2 + $TTS_{4.5}$) at 4000 Hz of the 4 subjects with the most TTS from 24 hr and 48 hr exposure of pink noise at 85 dBA.

The noise used in the 24-48 hr exposures was pink noise (the octave band Sound Pressure Levels were equal from 125 Hz to 4000 Hz). This noise was chosen to better approximate many typical environmental noises. The noises from jet aircraft, for instance, are reasonably well approximated by pink noise. For pink noise, Fig 5 shows the results of looking at the various audiometric frequencies. The greatest effect is at 4000 Hz while below 2000 Hz not much TTS occurs. The growth and recovery at 2000 Hz from the 85 dBA pink noise exposure is very consistent with the growth and recovery of TTS from an 1/3 octave band of noise centered at 1000 Hz as indicated in Fig 1.

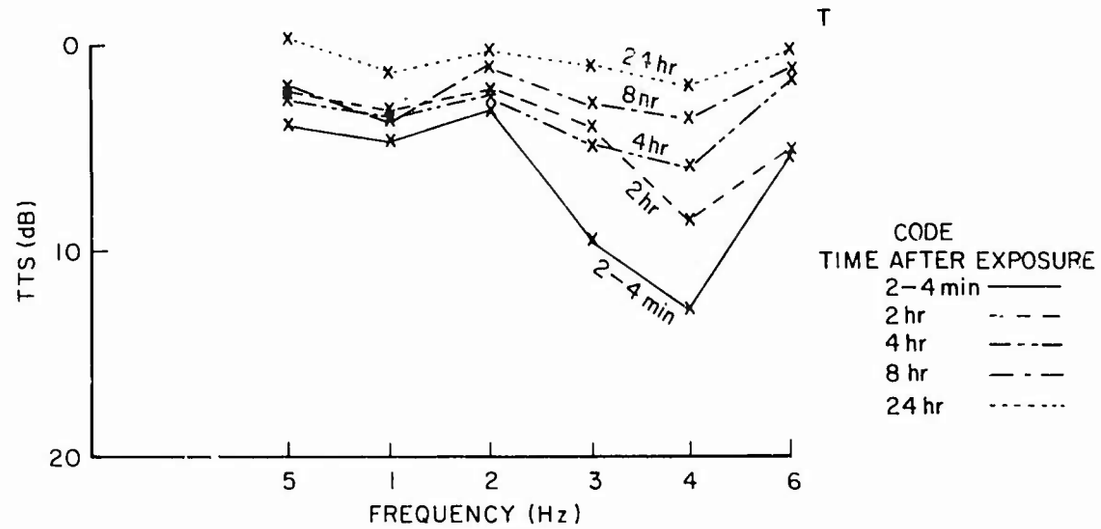

Figure 5. Recovery of Asymptotic TTS from a 48 hr noise exposure of pink noise at 85 dBA.

There are several questions still unresolved at this time. One of these is the location of the threshold of Asymptotic TTS for typical noises, such as pink noise. Such a threshold should serve as the lower bound at which noise can damage the ear at all. We say this rather positively because if a noise does not cause a TTS, it certainly cannot cause a PTS. Mills has proposed that such a point might be below 65 dB for an octave band centered at 4000 Hz (1, 12). This would mean for pink noise that approximately 72 dBA should be such a threshold. From our 24-48 hr studies, the Asymptotic TTS_2 for 85 dBA pink noise was 12 dB. Using the formulation proposed by Carder and Miller (4) and supported by Mills (12) that ATS = 1.7(OBL-C), the threshold level (C) would be estimated as 78 dBA. However, the results that we obtained in Figure 1 do not completely support the fact that average ATS grows 1.7 dB for each dB the noise is above some critical level C. In fact, the average ATS is 7 dB for the OBL of 80 dB, 10 dB for 85 dB, and 13 dB for 90 dB. This growth of ATS is better predicted by a formula ATS = .6(OBL-69). This slope of .6 is significantly different than the 1.7 reported by Carder and Miller. Now the slope of 1.7 dB is based on

chinchilla data except for the one individual human subject of Mills. If we look at individuals, the 12 subjects (of the study referenced in Fig 1) had slopes that varied from 0.2 to 2. In Melnick's studies (5), such variability was also apparent for 16 hr exposures of 85 dB, 90 dB and 95 dB of a noise spectrum predominately between 300 Hz to 800 Hz. Average growth of TTS for 5 subjects of Melnick varied from a slope of approximately 1 for $TTS_{3.5}$ at 750 Hz to a slope of 1.3 for TTS_5 for 1500 Hz. What this conflicting data indicates is that more research is needed if the threshold value of C in the above formulas is to be found. Furthermore, this threshold value should be found experimentally by lowering the levels of 24 hr exposures until no TTS occurs. The estimation of this threshold level by use of ATS = B(OBL-C) does not seem warranted because of the difference in the values of the slope B reported by various researchers. This is why we are currently planning to perform 24 hr studies for exposure levels of 65 to 80 dB within the next year.

Another question of practical interest is at what level should any long duration exposure be avoided, regardless of the amount of recovery time that can be allowed. This maximum level should be below where a person can receive a Permanent Threshold Shift from a single noise exposure. This level cannot be found experimentally, for obvious reasons. Therefore, it is estimated that this is the noise level that will cause more than a 40 dB Asymptotic TTS. From the data we have from the 85 dBA exposures and by assuming that the amount of ATS is normally distributed, we can predict, using either the 24 hr data or the 48 hr data, that less than 1 person in 200 will have more than 30 dB ATS. Considering the growth of ATS versus level as 10 dB for a 5 dB increase in level (B=2), which was the greatest rate of any subject, then 90 dB would cause an ATS more than 40 dB in well less than 1 person in 200. It should be emphasized here that we expect most ATS over 40 dB to recover even though described as potentially dangerous. At any rate, 90 dB is visualized as being acceptable given adequate recovery time. Somewhat higher levels may also be safe, but a conservative approach is to treat them as potentially dangerous.

One final problem with research in Asymptotic TTS is the effect of interruptions of the noise exposure on the growth of TTS. Since in the practical world, interruptions of noise exposure are hard to avoid, this study is needed to better apply long duration studies to real life situations. Therefore, our current research effort will also be directed to determine the effect on Asymptotic TTS of various interruption patterns. To illustrate the problem of preventing interruptions, in the studies we perform, we have found it necessary to have a person constantly watch sleeping subjects so that they do not lie on the ear which is receiving the exposure. Thus even in our controlled experiments, uninterrupted long duration exposures are difficult to provide.

Guidelines for Long Duration Exposures

As stated in the foreword, our proposal follows as to how long duration exposure may best be viewed for hearing conservation purposes from an operational standpoint, taking into consideration what we do not know about such exposures. First, long duration exposures should be defined as noise exposures above 65 dB that last for more than 16 hrs. Second, long duration exposure to noise levels above 90 dB should be avoided. Asymptotic TTS values in excess of 40 dB may be such that permanent changes for some individuals may occur from a single exposure. Third, long duration exposures to levels between 80 and 90 dBA should be treated as potentially hazardous. The suggested rule of thumb is to provide a recovery in relative quiet (<65 dBA) that is at least as long as the exposure duration. Thus a person who experiences a 3 day mission at 85 dBA should be allowed at least 3 days of quiet before the next mission. Fourth, exposures between 70 and 80 dBA are in a transition zone in which it is probably safe to allow rest for 1/2 of the exposure duration. Fifth, exposures less than 70 dBA are probably safe for all individuals, regardless of availability of recovery in levels less than 65 dB. There are no sharp breaks between the effects of exposure levels such as 79 dB and 80 dB, so some interpolation might be suggested.

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DISCUSSION

Q. (Melnick) One of your figures showed that the growth of noise induced temporary threshold shift starts after one hour.

A. (Johnson) We find that we get a growth of noise induced temporary threshold shift rather rapidly.

Q. (Melnick) Do you get any growth between 0 and 1 hour?

A. (Johnson) We do not test during the first hour so we have no way of knowing.

Q. (Melnick) This figure implies that there is no threshold shift during the first hour of exposure.

A. (Johnson) It is an idealized figure. The curve should not have been extended down to 0 hours.

THE INCIDENCE OF TEMPORARY AND PERMANENT HEARING LOSS AMONG AIRCREWS EXPOSED TO LONG-DURATION NOISE IN MARITIME PATROL AIRCRAFT*

S.E. FORSHAW
Behavioural Sciences Division
Defence and Civil Institute of Environmental Medicine
P.O. Box 2000
Downsview, Ontario, M3M 3B9
Canada

SUMMARY

The CP-107 Argus has been in operation with the Canadian Forces since 1957 as a long-range maritime patrol aircraft. The endurance capability of the aircraft is at least 24 hours at reconnaissance altitudes and speeds.

Flight durations from 12 to 20 hours occur routinely, during which ambient noise levels at various crew and rest stations range from 90 to 99 dBA. An assessment of crew and operational problems arising from long-duration flights in the Argus has shown that about half of the crew sustain temporary threshold shifts in excess of levels considered to be acceptable for long-term exposure.

A study of the hearing levels of 223 pilots, navigators and flight engineers with career flying times in the Argus ranging from 2500 to 10,000 hours suggests, however, that repeated long-duration noise exposure, as experienced in the aircraft, is not any more deleterious to hearing thresholds than is repeated exposure, at approximately equivalent intensity levels, in short- and medium-range aircraft.

INTRODUCTION

Although there is almost no published information on the effects of long-duration exposures to noise upon the human auditory system, it is clear that the resulting temporary threshold shifts (TTS) become asymptotic with time beyond certain duration limits. The study of Mills et al (1)** suggests that for man, the time required for a TTS to reach its asymptote is somewhere between four and 12 hours, and the subsequent return to the pre-exposure threshold takes from three to six days.

The long-term consequences of repeated noise exposures that produce asymptotic levels of TTS are not known. Their investigation is vital, however, to our understanding of the mechanisms occuring in the cochlea during intense stimulations that produce temporary and permanent threshold shifts (PTS).

It has been demonstrated that a lack of PTS after exposure to noise does not assure an absence of damage in the inner ear. Audiometric and histological examinations of chinchillae, by Bohne et al (2), for example, have shown considerable outer hair cell depletion from exposure to long-duration steady-state noise, even though pre-exposure thresholds returned after cessation of the exposure. Henderson et al (3) report the occurence of even larger lesions of the outer hair cells with small losses of the inner cells in the chinchilla exposed to impulse noise, without accompanying elevations in pure-tone threshold levels.

This raises a question of experimental ethics, for surely an investigator should limit the magnitude of induced TTS in human subjects, (and thereby perhaps the usefulness of an experiment) if he is to minimize the risk of permanent damage to the chochlea. If so, the study of hearing-loss incidence in certain populations, who by the nature of their occupations sustain intense or long duration noise exposures, may be an alternative method of research.

NOISE EXPOSURE IN CANADIAN FORCES LONG-RANGE MARITIME PATROL AIRCRAFT

The CP-107 Argus, a modified Bristol Britannia built by Canadair Limited of Montreal, has been in operation with the Canadian Forces (CF) since 1957 as a long-range maritime patrol aircraft. It is powered by four Wright R-3350 turbo-compound piston engines that provide an endurance capability of at least 24 hours at reconnaissance altitudes and speeds***.

Flight durations from 12 to 20 hours occur routinely, during which periods ambient noise levels at various crew and rest stations range from 90 to 99 dBA at normal cruise (see Figure 1 and Table I). It is difficult, however, to ascertain actual noise exposures in such an environment.

One is never certain, for example, of the effectiveness of a flight helmet or headset in attenuating noise, particularly when the noise (as in the Argus) is predominantly low frequency. The noise reduction

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* DCIEM Research Paper 75-RP-1073.

** One subject was exposed on two occasions to octave-band noise centered at 500 Hz. The first exposure lasted for 48 hours to a sound pressure level of 81.5 dB, the second for 29.5 hours to 92.5 dB. The asymptotic levels of the resulting TTS were 10.5 and 27.5 dB respectively.

*** The aircraft's endurance record is 31 hours.

attributed to the CF standard-issue flight helmet* is valid only if it is correctly sized for the wearer and optimally adjusted (a condition not always realized), and is worn with its chin strap reasonably tight. If the helmet is fitted with the smallest of the three sizes of earcups available, its attenuation will be further reduced by 5 to 7 dB at low frequencies. It has been shown, moreover, that the low-frequency noise protection of an otherwise effective helmet or headset is reduced by 3 to 8 dB with standard rim glasses (4), and by 3 to 5 dB with standard pattern aviation spectacles (5).

Estimating noise exposures in this aircraft is further complicated by the fact that Argus crews can spend up to one-third of the flight time in the galley and rest areas during long-range reconnaissance patrols. They do not always wear hearing protection during these periods.

SHORT-TERM EFFECTS OF LONG-DURATION ARGUS NOISE EXPOSURE

As part of a study (6) carried out to assess crew and operational problems resulting from long-duration flight in the Argus, the author measured the TTS sustained by a 15-man crew during a 16-hour flight in the aircraft. Pre-flight hearing thresholds were obtained at the crew's home base prior to take-off on the first flight of the mission**, and within 90 minutes of landing after the second flight (see Table II).

Since the pre- and post-flight hearing thresholds had to be measured at two bases using different audiometers, differences of ± 10 dB between the two are not considered significant. Three of the crew, one (No. 5, Table II) with a pre-flight bilateral loss of 20 dB at the four test frequencies, and two (Nos. 2 and 8) with pre-flight unilateral losses of at least 25 dB at 4 and 6 kHz, were considered to have sufficiently high levels of PTS to preclude significant TTS in the affected ears. A fourth crewman (No. 4) developed serous otitis media in his left ear, thus preventing meaningful post-flight audiometry.

Of the remaining 25 ears, 13 were observed with significant TTS: nine with TTS up to 20 dB at one of the four test frequencies, two with up to 20 dB at two of the four test frequencies, and two with TTS greater than 20 dB at at least two of the four test frequencies.

Alternately, 12 of the 25 previously defined 'susceptible' ears were observed with post-flight thresholds in excess of the levels of PTS considered by CHABA (7) to be acceptable after many years of noise exposure***. It is interesting to note that four of the six crewmen (Nos. 4, 6, 8, 12, 14 and 15) who spent at least three of the last four hours of the second flight in the most intense noise areas of the aircraft (in the plane of the aircraft's propellers), exhibited post-flight hearing thresholds (three bilateral, one unilateral****) exceeding the CHABA acceptable criterion.

At the time of this study, the author was not concerned whether or not the observed TTSs were asymptotic. Hence, the rate of recovery of pre exposure thresholds was not monitored. All that can be said regarding this point is that crew thresholds had returned to their pre-flight levels within 120 hours of completion of the third flight of the mission.

LONG-TERM EFFECTS OF ARGUS NOISE EXPOSURE

More conclusive information on the consequences of repeated long-duration noise exposure may perhaps be obtained by examining the incidence of hearing loss in individuals who sustain such exposure.

Accordingly, two populations of CF aircrew (pilots, navigators, flight engineers) are considered: those whose career flying experience has been mainly in the Argus (individuals whose occupational noise exposure has been typically long duration), and as a control group, those whose career flying experience has been entirely in short- or medium-range piston-engine and/or turbo-prop aircraft. These aircraft, and their octave-band noise levels at normal cruise (8, 9, 10, 11), are shown in Table III.

The hearing levels of CF personnel are classified for the purposes of enlistment, career assignment, and medical reassessment by the categories listed in Table IV. The percentages of personnel in the two aircrew populations, having career flying times from 2500 to 4000 hours, 4000 to 5500 hours, and greater than 5500 hours, are shown in Table V. The respective population-group sizes are n = 109, 61 and 53, and 86, 47 and 57 as shown.

The effect of accumulative flying time upon hearing loss, defined herein by hearing category H2, H3 or H4, can be seen more clearly in Figure 2 where the percentage occurance of H1 category in the two

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* The calculated sound pressure levels at the entrance to the ear canals of the pilot, flight engineer, and routine navigator wearing CF standard-issue flight helmets, based on (1) the noise levels given for these crew positions in Table I and (2) the attenuation provided by the flight helmet (optimally fitted) for the 83rd percentile of the population, are 79, 81 and 92 dBA respectively.

** The crew of one Argus aircraft was observed during a routine northern patrol totalling 49.3 hours, divided into three flights of approximately equal duration, over a 104-hour period.

*** The CHABA damage-risk criterion for steady-state noise (7) considers a permanent hearing loss to be acceptable, after many years of noise exposure, if it does not exceed 10 dB at or below 1000 Hz, 15 dB at 2000 Hz, and 20 dB at or above 3000 Hz.

**** Post-flight audiometry was not conducted in the left ear of crewman No. 4 because of serous otitis media.

aircrew populations is shown as a function of career flying time. Differences in the proportions of HI category among Argus and short- and medium-range crews are not significant ($p>.05$)*. Hence, although a real difference in the incidence of hearing loss in the two populations may indeed exist, a difference is not evident from these data.

There are a number of factors, of course, (e.g., presbycusis, non-occupational noise exposure certain illnesses and medications) that can interact with effects that may result from occupational noise exposure. Moreover, such interactions are likely to affect population hearing levels more significantly with increased age.

On the other hand, Pierson and Barren (12) suggest that experienced patrol aircraft crews may in fact represent a select population of 'noise-resistant' ears, and that hearing loss eliminates the overly susceptible individuals before they can accrue many flying hours. Whether or not this has been a significant factor in the above CF aircrew population survey has not been established.

It is, nevertheless, possible that any extraordinary effect that repeated long-duration noise exposure may have upon the hearing levels of maritime patrol aircraft crews, particularly when career flying time exceeds 5500 hours, may be masked by other contributing factors.

It is acknowledged, moreover, that the difficulty encountered herein in defining more precisely the operational noise exposure levels and durations of the two aircrew populations (a problem perhaps inherent in most epidemiological studies of noise), and the manner in which hearing losses were necessarily categorized, probably precludes the detection of any subtle difference in hearing deterioration in the two populations.

Given these limitations, one may state simply that there is no evidence in these data to suggest that repeated long-duration noise exposure, as experienced in long-range maritime patrol aircraft, is any more deleterious to hearing threshold levels than is repeated exposure, at approximately equivalent intensity levels, in short- and medium-range aircraft.

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* For the three classifications of career flying time shown in Figure 1 (2500-4000 hours, 4000-5500 hours, and 5500 or more hours), χ^2 = .042, 1.45 and .0032 respectively (using Yates' correction for continuity).

TABLES AND FIGURES

TABLE I

THE ARGUS AIRCRAFT
OCTAVE-BAND SOUND PRESSURE LEVELS

	OCTAVE-BAND CENTRE FREQUENCY SPLs dB									OVERALL SPLs	
MEASUREMENT LOCATION, ENGINE SPEED, ETC.	31.5 Hz	63 Hz	125 Hz	250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz	dBC	dBA
NOSE OBSERVER, FAN ON, 2100 RPM.	98	96	93	97	93	86	84	72	67	104	93
PILOT, A/C ON, 2200 RPM.	94	101	102	93	92	90	82	82	71	106	95
FLIGHT ENGINEER, 2320 RPM.	92	102	104	96	90	85	74	68	62	107	92
ROUTINE NAVIGATOR, 2320 RPM.	99	106	105	111	97	88	77	69	58	113	99
RADIO OPERATOR, 2320 RPM.	96	110	108	104	93	82	73	65	58	113	98
GALLEY, AFT SEAT, CENTRE OF TABLE, 2200 RPM.	101	107	105	100	87	77	68	60	53	111	94
BUNK AREA, AFT STBD, 2320 RPM.	97	102	99	97	86	76	67	60	52	106	88
TACTICAL NAVIGATOR, 2320 RPM.	94	100	101	96	88	75	65	57	50	106	93
MAD OPERATOR, 2320 RPM.	92	100	98	99	86	76	66	60	52	105	90
BEAM SEARCH STATION PORT, 2320 RPM.	96	104	95	92	86	82	73	68	60	106	99

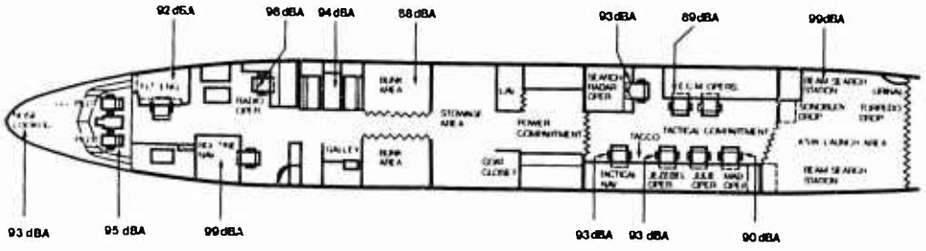

Figure 1: Plan view of the Argus aircraft showing the crew work and rest areas and overall A-weighted sound pressure levels.

TABLE II

ARGUS – CREW NOISE EXPOSURE HISTORIES AND HEARING THRESHOLD DATA

NUMBER	CREW POSITION	AUDIOMETRIC TEST TIME	HEARING THRESHOLD LEFT EAR				HEARING THRESHOLD RIGHT EAR				AGE	CAREER FLYING HOURS (SEE FOOTNOTE FOR LEGEND)
			2KHZ	3KHZ	4KHZ	6KHZ	2KHZ	3KHZ	4KHZ	6KHZ		
1	PILOT	PRE-FLIGHT	5	0	20	10	0	0	0	10	37	250(HAR), 75(NOR), 4500(DAK), 1300(LAN), 2200(BEE), 1700(ARG).
		POST-FLIGHT	15	20	20	25	5	5	5	10		
2	PILOT	PRE-FLIGHT	0	10	35	25	0	10	5	10	37	5200(NEP, ORN), 1500 (ARG)
		POST-FLIGHT	5	20	25	25	0	15	10	25		
3	PILOT	PRE-FLIGHT	0	5	10	15	0	0	0	0	26	350(EXP, TUT), 1100(DAK), 2000(ARG).
		POST-FLIGHT	10	10	15	25	0	0	0	10		
4	NAVIGATOR	PRE-FLIGHT	10	10	25	15	5	10	25	25	39	1800(NEP), 200(H44), 100(OTT), 300(DAK), 200(EXP), 600(LAN), 1000(ALB), 1200(ARG).
		POST-FLIGHT					20	30	35	35		
5	NAVIGATOR	PRE-FLIGHT	20	20	20	20	20	20	20	20	30	300(EXP,DAK,NEP), 3100(ARG).
		POST-FLIGHT	20	25	20	25	20	20	20	25		
6	NAVIGATOR	PRE-FLIGHT	10	15	5	5	0	5	10	10	38	2200(BOX), 1000(DAK), 2200(YUK), 2700(NOR), 1200(ARG).
		POST-FLIGHT	10	15	10	20	0	5	5	15		
7	NAVIGATOR	PRE-FLIGHT	0	0	0	0	0	0	0	0	23	200(DAK), 500(ARG).
		POST-FLIGHT	0	0	10	10	0	0	0	5		
8	OBSERVER	PRE-FLIGHT	0	10	40	50	0	0	10	10	25	110(DAK), 1690(ARG)
		POST-FLIGHT	0	15	30	40	0	5	10	15		
9	OBSERVER	PRE-FLIGHT	5	5	10	10	0	0	10	20	26	1000(TRA), 900(ARG).
		POST-FLIGHT	5	10	15	25	0	0	5	25		
10	OBSERVER	PRE-FLIGHT	0	0	3	5	5	0	10	0	35	40 (DAK), 1200(ARG).
		POST-FLIGHT	10	20	40	20	10	5	20	15		
11	OBSERVER	PRE-FLIGHT	0	0	0	5	0	0	10	5	27	50(DAK), 1400(ARG).
		POST-FLIGHT	5	10	10	20	5	5	10	20		
12	OBSERVER	PRE-FLIGHT	0	15	25	15	0	5	0	35	29	400(ARG).
		POST-FLIGHT	15	40	45	35	0	35	30	40		
13	OBSERVER	PRE-FLIGHT	5	0	10	10	0	0	10	5	39	2000(NEP, SHA), 2500(ARG)
		POST-FLIGHT	10	0	10	5	0	5	15	20		
14	FLIGHT ENGINEER	PRE-FLIGHT	0	10	15	10	0	10	20	25	38	600(DAK, EXP) 100(H34), 88(NEP), 3400(ARG)
		POST-FLIGHT	0	25	25	10	0	20	30	25		
15	FLIGHT ENGINEER	PRE-FLIGHT	0	5	25	15	0	10	20	15	39	4390(NOR), 4300 (DAK), 800(ARG)
		POST-FLIGHT	0	10	25	10	5	15	35	20		

ALB – ALBATROS, ARG – ARGUS, BEE – BEECHCRAFT, DAK DAKOTA, EXP – EXPEDITER, HAR – HARVARD, H34 – H34, H44 H44, LAN – LANCASTER, NEP – NEPTUNE, NOR – NORTHSTAR, ORN ORION, OTT – OTTER, SHA – SHACKLETON, TRA TRACKER, TUT TUTOR, YUK – YUKON.

TABLE III

NOISE LEVELS IN VARIOUS AIRCRAFT, NORMAL CRUISE CONDITIONS, FLOWN BY THE CANADIAN FORCES DURING THE LAST 25 YEARS

AIRCRAFT	OCTAVE-BAND CENTRE FREQUENCY SPLs dB									OVERALL SPLs	
	31.5 Hz	63 Hz	125 Hz	250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz	dBC	dBA
C47 DAKOTA – Pilot		103	103	95	90	83	79	72	65	107	93
– Navigator		103	106	107	101	93	82	76		111	102
C–119 PACKET – Pilot		107	100	91	88	89	86	84	83	108	94
– Navigator		104	103	91	88	87	84	81	82	107	93
P2V–7 NEPTUNE – Pilot		96	96	95	85	81	76	72		101	89
– Navigator		96	97	91	86	79	75	72	69	100	88
CC – 115 BUFFALO – Pilot	92	95	97	95	92	90	85	83	81	102	95
– Navigator	105	108	104	94	96	92	85	81	74	111	97
CC–109 COSMOPOLITAN – Pit	83	105	90	91	90	88	84	76	68	106	92
C–45 EXPEDITOR – Pilot		106	107	100	98	90	79	74		110	98
CS2F TRACKER – Pilot		91	93	89	82	80	78	77		97	88
HARVARD – Pilot		104	108	109	102	94	88	86	90	113	104

TABLE IV

CANADIAN FORCES HEARING STANDARDS

CATEGORY	HEARING STANDARD[1]
H1	Hearing level not greater than 30 dB between 500 and 6000 Hz in both ears.
H2	Hearing level not greater than 30 dB between 500 and 3000 Hz in both ears.
H3	Hearing level not greater than 30 dB between 500 and 2000 Hz in the better ear.
H4	Hearing level not greater than 50 dB between 500 and 2000 Hz in the better ear.

[1]Hearing levels are relative to ISO reference threshold levels (1964).

TABLE V

THE DISTRIBUTION OF HEARING CATEGORIES AMONG CERTAIN CF FLIGHT PERSONNEL

	ARGUS CREWS			SHORT- AND MEDIUM-RANGE CREWS		
	CAREER FLYING TIME					
	2500-4000 HRS	4000-5500 HRS	>5500 HRS	2500-4000 HRS	4000-5500 HRS	>5500 HRS
H1	88.1	82.0	62.3	86.1	70.2	70.2
H2	11.0	16.4	26.4	12.9	14.9	21.1
H3	0.9	1.6	11.3	1.0	11.8	7.2
H4					2.1	1.5
n	109	61	53	86	47	57

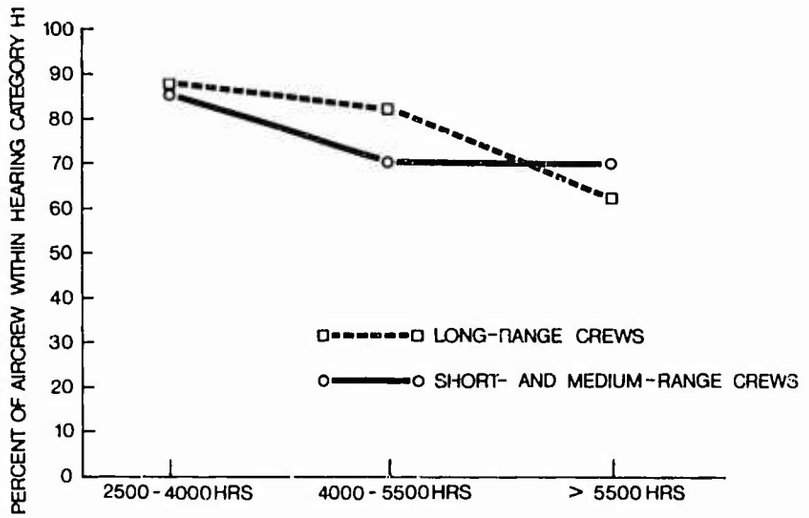

Figure 2: Percentage distribution of long-range (Argus) and short- and medium-range crews within CF hearing category H1, as a function of career flying hours.

DISCUSSION

Q. (von Gierke) In your next to last figure you showed the percentage of air crews within hearing category H-1 distributed over the accumulated flying hours and showed that this function is the same for the crews of long range aircraft as it is for the crews flying short range aircraft. I wonder, does it take the same time to accumulate 5,000 hours flying experience for both these crews in terms of the number of years of exposure?

A. (Forshaw) As of this moment I don't have the precise answer to your question. We are checking into this problem currently. We do know that in the last two or three years Argus crews had been flying between 60 and 80 hours a month. It is very difficult to put such a number on the other population because they include many different kinds of aircraft. Personally, I doubt very much if the flying time per month is grossly different between the two groups but this is all that I can say for now.

Q. (Johnson) That last figure shows that the hearing of the normal population is actually a little bit worse than the hearing of your pilots in either group. One might conclude from this that the noise exposure from flying has no effect on hearing. In fact, though, don't you have a screening effect in that you pick people with good hearing to become pilots? This is what we do in the United States.

A. (Forshaw) This is correct. The flight populations are a selected group whereas our so-called normals in that last figure had no prior screening at all for their hearing.

Q. (Johnson) Where is the figure published?

A. (Forshaw) This figure will be published in the proceedings of a symposium held in Canberra, Australia this year.

PSYCHO-PHYSICAL PERFORMANCE OF AIR FORCE TECHNICIANS AFTER LONG DURATION NOISE EXPOSURE

by

Col. Cesare A. Ramacci, IAF MC
Lt. Col. Paolo Rota, IAF MC
Centro di Studi e Ricerche di Medicina Aeronautica
e Spaziale
via P. Gobetti 2A
Roma, 00185, Italy

SUMMARY

A few psychological and psycho-physiological tests were carried out (Toulouse Piéron test, flicker fusion test, reaction time) in 20 Air Force technicians, on duty at aircraft maintenance and flight line, exposed to high level noise.

Work conditions considered were: (i) continuous exposition to noise of about 120 db, for 1 hour and half;(ii)continuous exposition for 5 hours to 60-80 db noise, with transient increases up to 90-115 db. The technicians used,when necessary, individual or collective protection.

The tests, carried out before and after noise exposure, did not show significant changes of performance.

PREFACE

Extended exposure to high levels noise is a relevant problem for the Air Force personnel, and its dangerous effects were studied by a large number of reseachers. Apart from the effects on hearing, that we will not consider in this paper, a few studies have shown, yet with contrasting evaluations, possible non acoustic effects (biochemical, functional and psychological) in subjects exposed to intense noise, mainly when combined with vibration (1,2,3,4).

In order to demonstrate these effects in the actual operational situations of the Air Force, we have examined the psycho-physiological behaviour of technicians, at the start and the end of their work shifts, in an Air Force base, where they were exposed to noise of different intensities.

ENVIRONMENTAL CONDITIONS AND TESTS RESULTS

The study has been carried out in summertime, on the personnel of an Air Force base, on duty at jet aircraft maintenance and flight line, in occasion of the morning shifts, 07.30 to 13.00, with normal work schedule.

Twenty subjects were studied, all experienced in their service, divided into three groups, each of them being charged with specific tasks. The subjects were exposed,both due to the work itself and the proximity of the runway, to noise of different levels (sound levels, measured with Elit 905 phonometer, are reported below).

A first group of 10 individuals, on duty at the flight line, was exposed to basic noise of 80db, C filter, with transient increases between 90 and 110 db, and for some specific task up to 115 db, always with C filter.

The second group, of 5 individuals, was in service in a workshop for aircraft maintenance and repair, and exposed to basic noise of 60db,C filter, with peaks of 100-110 db, C filter.

The third group,of 5 subjects , besides working in the shop, as the second group, was also in charge at the engine test station, being exposed to noise between 100 and 135 db, C filter, for 1 hour and half.

As far as the work environment is concerned, we noticed also the contemporary presence of vibration, that were not measured. During the study, sky was clear, air temperature between 20 and 25 C, wind of about 10 knots. All the personnel donned, when necessary, the anti-noise head set, or accomplished his task in protective booths.

All the subjects, at the start and the end of their shift, were submitted to flicker

fusion test, reaction time determination to visual stimuli, and cancellation test of Toulouse Piéron. The results of the study, as averages and standard deviations, are reported in table I, both for the three groups separately and all the subjects.

CONSIDERATIONS AND CONCLUSIONS

The results reported in table I show, for all the subjects, mild increase of reaction times and fusion frequencies, and improvement in the cancellation test, at the end of the work shift. We found also the same behaviour in all the three groups, separately considered, with the exception of individuals in service at the flight line, for the reaction time test.

Yet the changes found in the results of the tests carried out before and after the shifts, are always of mild value, and statistically not significant. On the other hand, the results of the tests carried out after the work shift, can possibly be affected by the learning process, due to the repetition of the tests.

On the whole, the data obtained in the tests carried out, don't put into evidence any different behaviour of the subjects, before and after their work shift, and are consistent with the behaviour of cathecolamines urinary excretion, studied in the same conditions (5).

In the evaluation of the results, that would fail to show valuable changes of performance, we must nevertheless notice that: all the subjects were adapted to their tasks; the tests, exploring only limited aspects of psychophysical activities, can not evidence transitory impairments during the work; the work schedule allowed for some recovery periods; the repetition of the test, as above said, can affect the results.

Therefore, without being able to exclude possible performance decrease and more severe fatigue of the Air Force techniciane, due to the accomplishment of their professional tasks in noisy environment, we feel that these possible effects should be, at least in the conditions we studied, of moderate importance and susceptible of quick recovery.

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TABLE I. Reaction time (hundredths of second), fusion frequency (flashes per minute), and Toulouse Piéron Test

	REACTION TIME							
	ENGINE TEST		WORKSHOP		FLIGHT LINE		ALL	
	$\bar{x}$	σ	$\bar{x}$	σ	$\bar{x}$	σ	$\bar{x}$	σ
BEFORE	24.9	4.83	26.1	5.34	26.4	5.61	25.9	5.35
AFTER	26.3	6.18	29.0	5.98	25.3	6.46	26.4	6.27

	TOULOUSE PIERON TEST							
	MARKED SIGNS				ERRORS + OMISSIONS			
	ENGINE TEST	WORK SHOP	FLIGHT LINE	ALL	ENGINE TEST	WORK SHOP	FLIGHT LINE	ALL
$\bar{x}$ BEFORE	110	109	105	107	15	16	20	17
$\bar{x}$ AFTER	112	112	115	114	13	13	10	11

	FUSION FREQUENCY			
	ENGINE TEST	WORK SHOP	FLIGHT LINE	ALL
$\bar{x}$ BEFORE	46.8	47.8	52.7	50.2
$\bar{x}$ AFTER	50.7	47.9	54.8	52.1

THE EFFECTS OF EAR PROTECTORS ON SOME AUTONOMIC RESPONSES TO AIRCRAFT- AND IMPULSIVE NOISE

by

G.R. FROEHLICH, Col., GAF, MC
German Air Force Institute of Aviation Medicine
Fürstenfeldbruck

INTRODUCTION

After extensive studies of the aural effects of noise, in recent years there has been an increasing interest in the non-auditory physiological effects of noise on men. The first investigators were primarily concerned with the effects of intense noise on the circulatory system. Here we encounter as the most reliable finding peripheral vasoconstriction together with more variable other cardiovascular changes. In conjunction with respiratory and endocrinological changes, all these effects are physiological responses within the frame of ergotropic mechanisms.
Since all these responses depend primarily on the intensity, duration and spectral character of noise, the wearing of ear protectors must decrease the physiological responses. We therefore have chosen as accustical stimuli an impulsive noise and jet aircraft noise together with three different types of ear protectors currently in use in the German Armed Forces (Fig. 1).

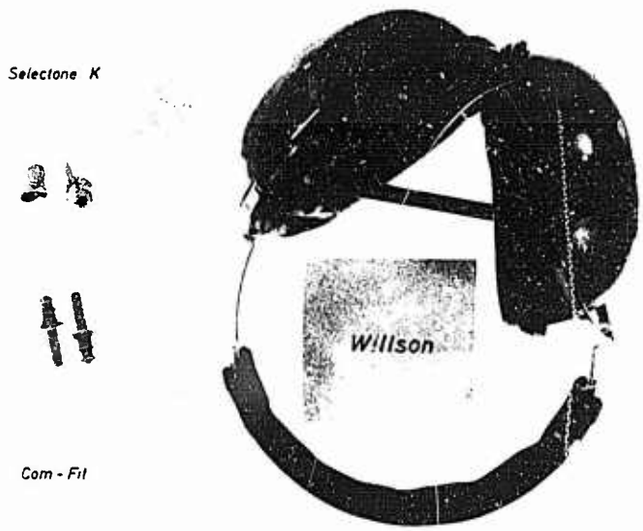

Fig. 1

The ear protectors described in the text.

METHODS

The SELECTONE K earplug contains two tiny holes, one connecting the inner cavity of the earplug with the outside air, the other with the air volume enclosed in the ear canal. Thus it acts as a two-section low-pass filter with low attenuation for the higher frequencies.

The COM-FIT earplug is a very efficient ear protector, providing excellent attenuation for all frequencies.

The WILLSON Sound Barrier Earmuff SB 258 is widely known and provides high attenuation already at 1000 Hz. The attenuation characteristics are shown in Fig. 2.

The impulsive noise was produced by discharging a children's pistol at a distance of 30 cm from the left ears of the normal hearing subjects, thereby producing 130 $\pm$ 2 dB(lin). In order to avoid permanent hearing damages, we dispensed with firing real pistols in the closed reverberating room. All four situations have been equally distributed to avoid habituation effects.

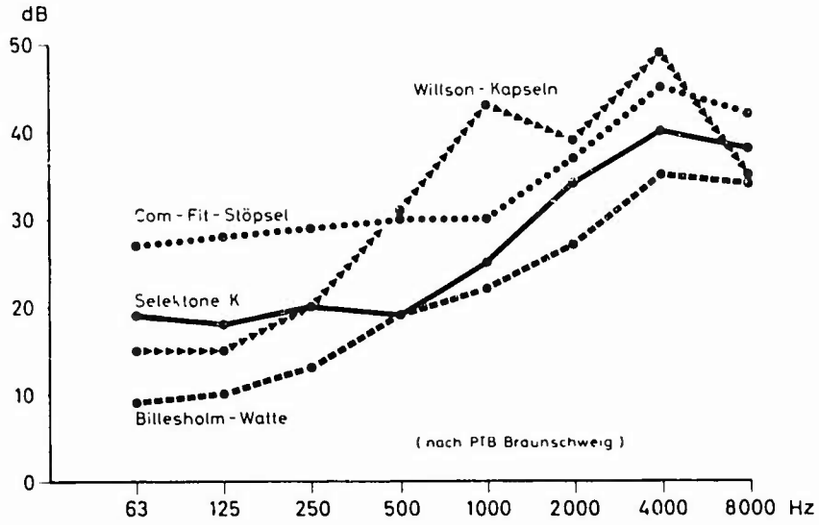

Fig. 2

Attenuation provided by the ear protectors

As typical aircraft noise we have used a 20 sec F-104(G) jet aircraft noise of 95 dB(lin) = 95 dB(A), as demonstrated in Fig. 3. Since there is a discrete frequency in the octave band centered at 1000 Hz, there is a very annoying, shrieking sound within a broadband noise. The noise levels and spectra in the subjects' ear canals were calculated by subtracting from the ambient noise level the attenuation values of the three ear protectors.

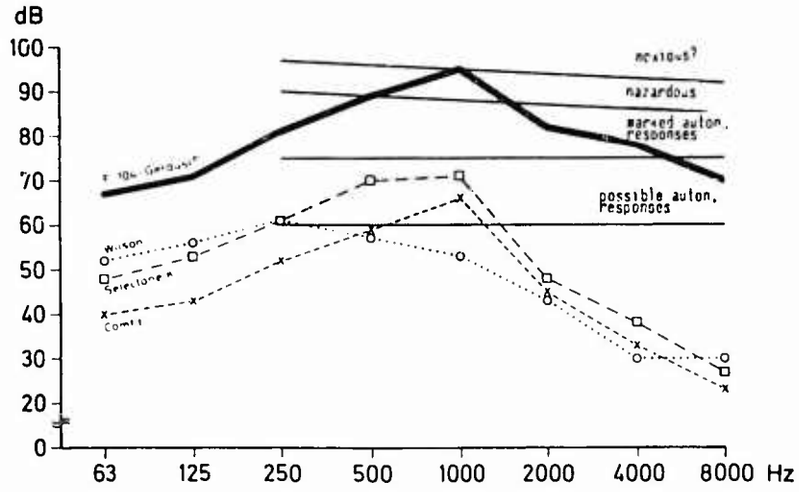

Fig. 3

Estimated noise rating for autonomic responses as proposed by JANSEN; spectrum of F-104 noise and its reduction by ear protectors.

The peripheral blood flow was determined by use of photoelectric transducers at the end-phalanx of the right middle finger. From these plethysmographic traces we could also compute the pulse frequency (Fig. 4)

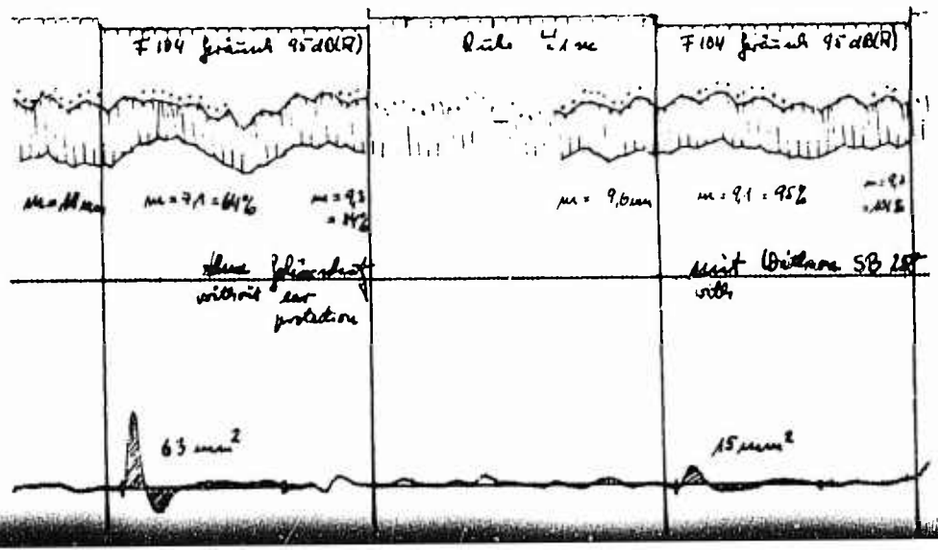

Fig. 4

Original traces of plethysmography and electrodermal response in F-104 noise without and with WILLSON earmuff

The electrodermal response (EDR) is the biphasic appearance of a voltage in response to an emotional stimulus. It reflects already minor changes in activity of the autonomic nervous system. The total plane of positive and negative phases were computed in mm^2.
All tests have been conducted in a room having normal reverberation at 22 - 24° Celsius between 14 - 1600 hours. In order to eliminate other sensory stimuli, the subjects had been blindfolded and possible random noises had been masked by a continous 50 dB(A) white noise.

RESULTS

1. The Effects of Ear Protectors on Peripheral Vasoconstriction Caused by a 20 sec Jet Noise (Fig.5)

Taking the average of the last 10 amplitudes before the sudden onset of noise as 100%, without ear protectors we have an amplitude reduction to 63,3% ± 12,6% as initial response and towards the end of the stimulation a recovery to 84,4% ± 13%. The amplitude reductions are considerably lower when ear protectors are used. With COM-FIT the amplitudes are reduced to 79% ± 12% and recover to 99%. The responses with the WILLSON earmuff are essentially the same: initial response 79% ± 10% and 97% ± 9% towards the end. The use of SELECTONE K offers less protection as is expressed by the more marked amplitude reductions to 72% ± 12% as initial response and a recovery to 94% ± 9% at the end of the 20 sec period. The differences of responses with and without the various ear protectors have been significant at the 0,001 level.

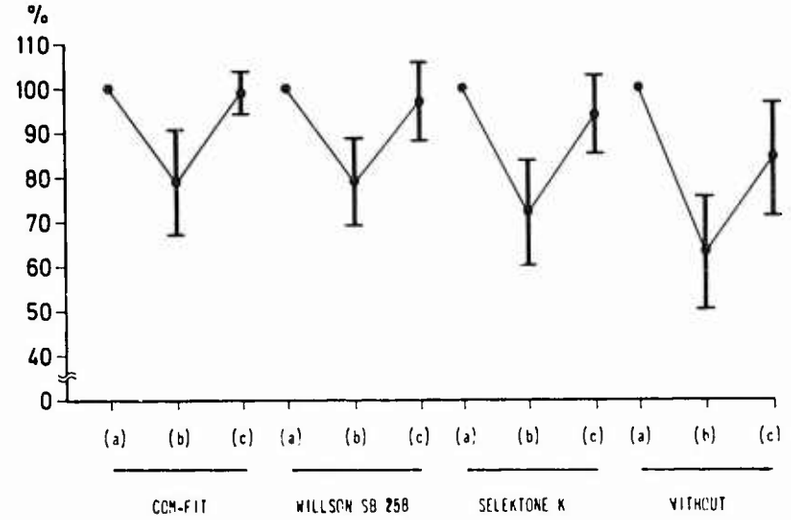

Fig. 5

Peripheral vasoconstriction caused by 95 dB(A) jet noise. (a) pre-exposure = 100%, (b) initial response and (c) recovery towards the end of exposure

2. Effects of Ear Protectors on Peripheral Vasoconstriction Caused by Impulsive Noise (Fig.6)

The initial responses after the 130 dB pistol shots without ear protectors show marked amplitude reductions to 62% ± 14%. With the use of COM-FIT there is only a small reduction to 86% ± 9% and with SELECTONE K to 79% ± 20%. In every intraindividual comparison, the protective

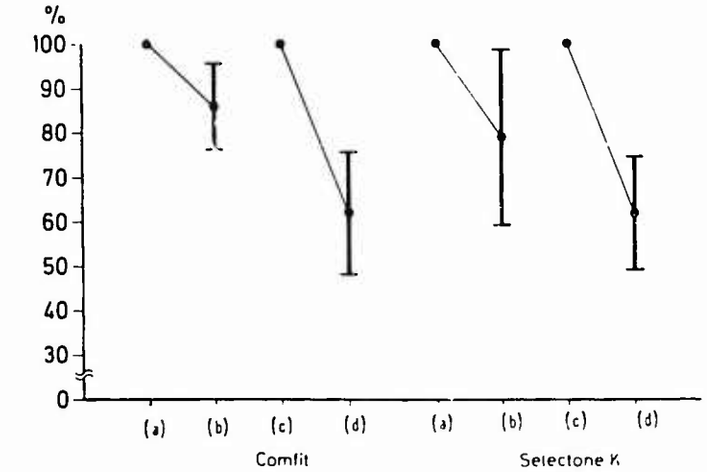

Fig. 6

Peripheral vasoconstriction caused by 13 dB impulsive noise.
(a)pre-exposure = 100%
(b)with ear protectors,
(c)pre-exposure and
(d)without ear protector

3. The Effects of Ear Protectors on the Electrodermal Response (EDR) (Fig. 7)

If we take the EDR evoked by the F-104 noise without ear protectors as 100%, there is a considerable decrease of these responses when ear protectors are used. With COM-FIT in the external ear canal, the EDR is reduced to 53% ± 26%, with WILLSON to 51% ± 31% and with SELECTONE to 67% ± 24%. Thus, the effectiveness of the WILLSON earmuff and COM-FIT earplug is the same, that of SELECTONE K somewhat less. When the 130 dB impulsive noise is presented, the COM-FIT earplug achieves an EDR reduction to 60% ± 25%, whereas SELECTONE K shows a reduction to 56% ± 28%. In this case , the SELECTONE K especially designed for protection against impulsive noise achieves the same effectiveness as COM-FIT, which otherwise has better protective properties.

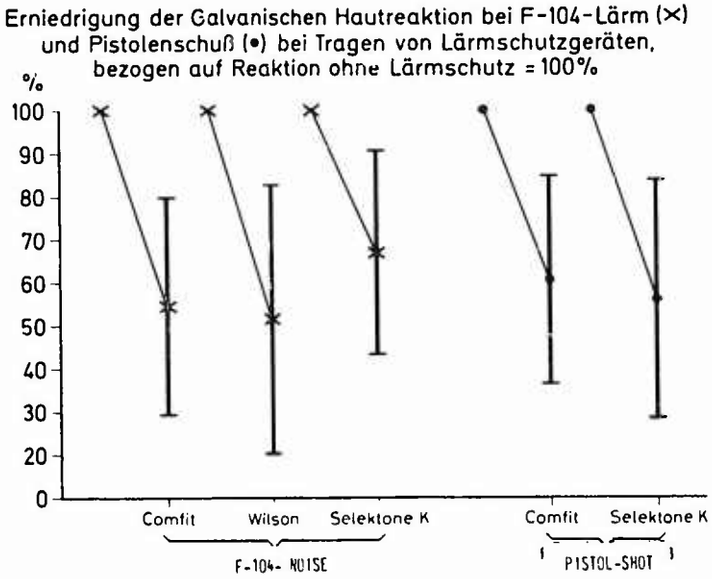

Fig. 7

Reduction of electrodermal response by wearing ear protectors. Responses without ear protection = 100%

4. Heart Rate

As already mentioned by several authors, there are no significant changes of the heart rate under the stress of noise, as can be seen in the following table:

		COM-FIT	WILLSON	SELECTONE K	WITHOUT
TABLE I:	Change of Heart Rates in % with 95 dB(A) noise	+ 1,8% ± 3,7%	+ 2,3% ± 3,7%	+ 2,4% ± 5,6%	- 1,7% ± 5,3%

		COM-FIT	WITHOUT	SELECTONE K	WITHOUT
TABLE II:	Change of Heart Rate with 130 dB Impulsive Noise	+ 0,1% ± 5,5%	- 0,45% ± 6,6%	+ 0,55% ± 3,3%	+ 1,65% ± 4,2%

5. Subjects' Assessment of Ear Protectors Against F-104 Noise

Immediately after the tests, each of the 25 subjects was asked to assess the effectiveness of the three different ear protectors and to establish an order of rank:

	ORDER OF RANK			
EAR PROTECTOR	1.	2.	3.	Average
SELECTONE K	2	3	20	2,7
COM-FIT	15	8	2	1,5
WILLSON	8	14	3	1,8

The COM-FIT ear plug has been assessed as the most effective ear protector, closely followed by the WILLSON earmuff. In accordance with the results of objective measurements, the SELECTONE K earplug is assessed as considerably less effective.

DISCUSSION

As shown by the frequency analysis in Fig. 3, marked autonomic responses had to be expected. They could be elicited for the peripheral blood flow as well as the electrodermal responses. Contrary to this, the heart rate showed no significant changes in terms of increase or decrease. The attenuation characteristics of the three ear protectors are quite different, so that at the eardrum of the subjects the intensities and qualities of the noise had been different too. By calculation, the protective effects of the WILLSON earmuff is best because the preponderant frequencies about 1000 Hz are attenuated most effectively. The second best is the COM-FIT earplug, whose attenuation at 1000 Hz is not as effective, but better in the lower frequency range. SELECTONE K has the least protective effect of all three in the lower and medium frequency range, whereas above 1000 Hz the attenuation properties are close for all three ear protectors. Thus, differencies in autonomic responses must be due to the different noise levels in the lower and medium frequency range.
While the exposure to F-104 noise without ear protectors is within the area of potential noxious effects (Fig. 3), the proper use of the ear protectors reduces the exposure to the area of only possible autonomic responses. All amplitude reductions as compared to pre-exposure amplitudes are significant at the 0,001 level as well as the differences without-with ear protectors. The protective effects of COM-FIT and WILLSON are essentially the same and both are significantly better than those of SELECTONE K. More important than the short-term initial effects are those towards the end of the 20 sec noise exposure period, since they indicate the constant response level for noise exposures of longer duration. Here we can safely state, that with COM-FIT and WILLSON the responses return to pre-exposure values during noise exposure, whereas with SELECTONE K a small vasoconstriction will still remain. The short-term initial response may be due to a certain startling effect at the rapid onset of noise. This opinion might be supported by the appearance of marked electrodermal response without ear protectors and a considerable decrease of this response by the use of WILLSON, COM-FIT and finally SELECTONE K. The standard deviations for the EDR are much larger, which makes the measurement of finger pulse amplitudes a more reliable parameter.

There was no fixed intraindividual relationship between the two test parameters inasmuch as under identical conditions strong responses in one parameter did not necessarily mean strong responses in the other one.

ACKNOWLEDGEMENT

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"INFLUENCE OF THE NOISE ON CATECHOLAMINE EXCRETION"

by
Lt.Col.Prof. G. PAOLUCCI, IAF, MC
Centro di Studi e Ricerche di Medicina Aeronautica e Spaziale
Via P. Gobetti, 2/a
00185 ROMA (Italy)

SUMMARY

Aim of the work was to know whether a few hours exposure to hazardous noises could act as a stressing factor and so able to give increase on catecholamine excretion.

The study was carried out on aviation specialists, daily exposed to high noises, fully protected against hearing damage by ear plugs; the exposed people were divided in two groups of ten subjects, eachone exposed to different noisy conditions:

- the one was exposed to continuous and steady noise of 120 dB for 1 hour and 1/2;
- the other to intermittent noise of 80-100 dB for 5 hours, with intervals between impulsive bursts of 20', lasting eachone only a few seconds.

The subjective tolerance was good and no disturbance or fatigue reactions appeared at the end of the exposure. Urinary catecholamine excretion was assayed the day before the test (in noiseless place) and the next one at the end of the exposure.

As the test values, compared with the blank, showed no changes in catecholamine release, it can be argued that, upon trained people, with hearing fully protected, noise might not act as a conventional stressor, at least at the same conditions of the present research.

INTRODUCTION AND AIM OF THE WORK

The exposure to high levels of noise for a certain duration, apart the auditory effects, can lead to a loss of working efficiency.

The damages of noise upon eardrum and hearing organ are not be taken into consideration in this paper, in which only endocrinological aspects are to.

Since the long exposure to noise, in most people can produce headache, drop on attention, loss of resistance, we intended to establish whether the noise could act as a stressing factor, able to give rise to increased output of catecholamines from adrenal gland.

METHOD

On 20 IAF ground specialists, usually employed in noisy tasks (wearing ear plugs during the job), this research was carried out. The people was divided in 2 groups of 10 persons, unhomogeneous for age and body size, which were exposed for different durations to different high noise conditions.

In particular, the exposure was executed in the following way in two different places :

- "A")- "engine test area": the workers remained exposed for 1 hour and 1/2; the noise was continuous and steady at level of about 120 dB;
- "B")- "take-off runway": the workers' exposure lasted 5 hours, but the noise was intermittent and lower; it arised at every F-104 take-off (one every 20') lasting only a few seconds and reaching about 80-100 dB.

In such people catecholamines were determined in a day off and after the noise exposure; the former was indicated "BLANK" and the latter "TEST". The analyses were executed according to BIO-RAD technique and the excretion values are reported in mcg/h.

As the "TEST" urinary specimen was collected at the end of exposure, and corresponded to the urine flown in bladder during all time of exposure, also urinary specimen of "BLANK" was collected for the same time, in the day before the test, in the same subjects resting in a noiseless place.

RESULTS

The following table shows the data obtained:

	Area "A" (continuous noise)	Area "B" (intermittent noise)
NOISE LEVEL (dB)	120	80-100
EXPOSURE DURATION (h)	1 1/2	5
BLANK (mcg/h)	4,82 ± 2,91	4,07 ± 4,74
TEST (mcg/h)	5,36 ± 3,03	4,74 ± 3,00

(We recall that normal catecholamine excretion, in other previous research determined, is 3-5 mcg/h)

At the end of the exposure all the subjects didn't feel tired and no fatigue symptoms appeared

CONCLUSIONS

This research has shown that no change happens on catecholamine excretion after a noise exposure for a few hours; all this seems to mean that noise can be heard without any adrenal gland response (perhaps human tolerance can be due to ear protection and noise training).

The catecholamine release was similar in all the workers but one, high duty responsabilities charged, whose "TEST" value was 4 times higher than "BLANK", confirming what we have already achieved in previous experiences, that psychic loads can cause catecholamine increase.

DISCUSSION

Q. I believe your method was concerned with the total measurement of catecholamine excretion. Have you obtained no effects by measuring total catecholamine excretion although the results were very variable? Is it possible that if you had measured differential catecholamine excretion such as the separate components of it that you would have found some changes? Also, subcortical-steriods, at least as far as animal experiments are concerned, are very sensitive to noise. Have there been experiments done in man or is it more appropriate to use corticosteriods in these types of estimations?

A. Catecholamine excretion is different from the cortico or the adreno cortico-steriod reaction because the catecholamine excretion is quickly responsive to stress while the cortico or 17-cortico-steriods produce a progressive reaction. Our experiments lasted for a short time and in this time no stress reaction was observed. We think that such stress might occur in humans to prolonged exposures at approximately 100 dB.

EFFECTS OF NOISE EXPOSURE

Robert W. Cantrell
Captain, Medical Corps, United States Navy
Chairman, Department of Otolaryngology
Naval Regional Medical Center
San Diego, California 92134

SUMMARY

It is known that noise can damage the inner ear, result in hearing loss, be a source of annoyance, disturb sleep, and interfere with speech. There is some evidence that it may adversely affect mental health, the cardiovascular system, basic biochemistry, and decrease work performance. This communication reviews the current knowledge of how intensity, duration and frequency composition of noise affects the auditory, annoyance, sleep and speech interference, psychological and sociological responses in man. Rising noise levels from ever-increasing sources have resulted in numerous complaints from the citizenry and have prompted the passage of various laws regulating noise and noise exposure. Health professionals are being consulted by legislative and regulating bodies for advice on allowable noise levels. It is important for all members of the health professions to be aware of the latest knowledge regarding the effects of noise exposure.

INTRODUCTION

In 50 A.D. Pliney the Elder reported in his Natural History that people living near one of the roaring cataracts of the Nile became hard of hearing. This is one of the earliest reports associating noise exposure and deafness. It has been confirmed many times since, that prolonged exposure to noise results in hearing loss. Noise is also known to be a source of annoyance, sleep disturbance and speech interference. Noise is reported to decrease mental and motor performance and may adversely affect mental health. This report will detail some of the effects of noise exposure.

Noise is defined simply as unwanted sound. This means that sound which is disagreeable, discordant, or which interferes with the reception of wanted sound becomes noise. Music played during the afternoon may be pleasant, but the same music played at 3:00 a.m. could be considered noise. A sound considered pleasant at one intensity becomes aversive and is perceived as noise if the intensity is increased markedly. Noise is thus a subjective experience with some people being bothered more by a particular sound than others either because of a different physiological state (e.g. headache, illness) or because of the information the sound conveys (e.g. the sound of a low-flying jet plane which raises the subjective feeling of a possible crash). It is this subjective variation which is difficult to express scientifically and which has given rise to many terms in attempting to quantify noise exposure.

AUDITORY EFFECTS

The auditory system is the most sensitive organ system in the body. Von Bekesy[1] has stated that the ear is sensitive enough to detect air molecules impinging on the tympanic membrane. Sound at 120 decibels (dB) sound pressure level (SPL), which is loud enough to cause discomfort, contains a power of only 1/10,000 of a watt (10^{-4} watts). This level is 1,000,000,000,000 times greater than the normal lower limits of audibility (10^{-16} watts).

Exposure to noise of sufficient intensity for sufficiently long periods of time results in a temporary increase of the threshold of audibility (Temporary Threshold Shift [TTS]). This loss usually can be regained in approximately 16 hours after the noise exposure terminates. Repeated noise exposures, very high intensities which cause large TTS, or prolonged exposure, can lead to a loss of hearing which is permanent (Noise-Induced Permanent Threshold Shift [NIPTS]).

When the organ of Corti of a patient with hearing loss due to noise exposure is examined histologically certain changes are noted. There is a decrease in the number of hair cells present, a decrease in the number of auditory neurons, and as the severity of the damage increases, there is complete collapse of the supporting structures of the organ of Corti, absence of the hair cells, and finally, complete degeneration of the organ of Corti. FIG. 1. represents a single cross-section through the organ of Corti with (a) normal, (b) partial injury, (c) severe injury and (d) total degeneration being represented. The organ of Corti is about 34 mm in length with an approximate total of 15,500 hair cells along its length. Sounds of different frequencies stimulate the basilar membrane at different areas, and one would expect the greatest damage at the point where activity is greatest. Essentially, this is true; however, for several reasons, sounds between 1000 and 4000 Hertz (Hz) produce the greatest distortion of the basilar membrane, and the greatest damage from noise occurs, at a point about 10 mm from the cochlear partition.

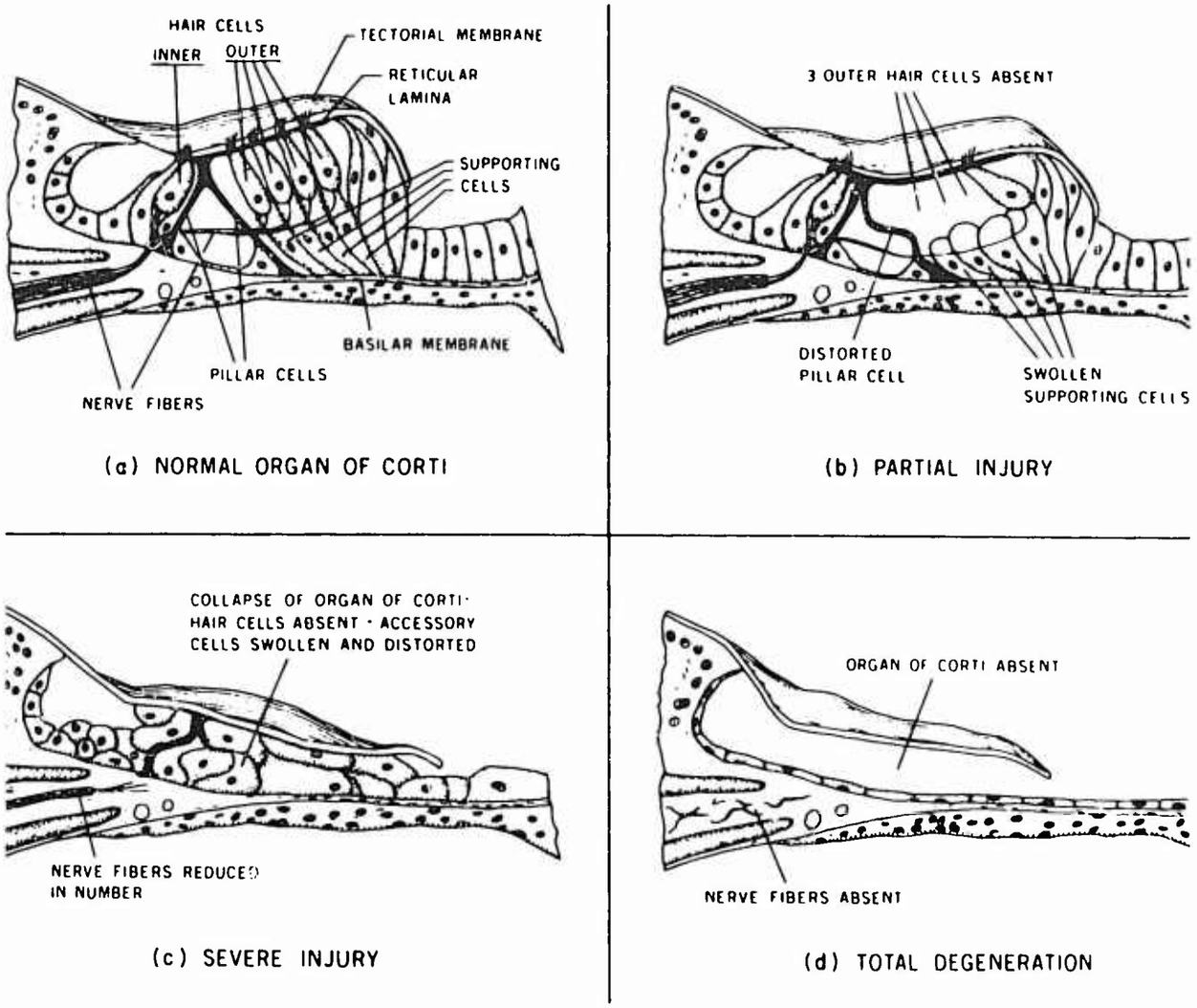

FIG. 1. Drawings of the human organ of Corti are shown that illustrate the normal state, panel a, and increasing degrees of noise-induced permanent injury, panels b, c, and d. (From Miller, J.D.: Effects of noise on people. J.Acoust.Soc.Am.Vol.56, No.3,September 1974, pp 729-764. (With permission of the author and the Journal of the Acoustical Society of America).

Very intense impulse noise such as gunfire or explosions are thought to vibrate the organ of Corti severely enough to disrupt the basilar membrane or shake the hair cells loose with resultant destruction. This is known as acoustic trauma.

Prolonged exposure to noise of 60-80 dBA (intensity as read on the A-scale of a sound level meter) for periods in excess of 8 hours will cause some temporary threshold shift (TTS) in most people, and increasing the intensity or prolonging the exposure will cause increased TTS and eventually noise-induced permanent threshold shift (NIPTS) which is irreversible.

It has been known for some time, that some people are more susceptible to damage from noise than are others. This individual susceptibility to noise-induced hearing loss further complicates regulation of noise exposure since one person may suffer adverse effects from a given noise exposure while another person may suffer little or no effects.

At any given sound pressure level low frequency noise is less damaging than high frequency noise. Noise from 1000-6000 Hz appears to cause the greatest TTS. Noise-induced hearing loss occurs initially at approximately 4000 Hz but can occur anywhere above 1000 Hz, and maximum impairment is usually at 5000 Hz.[2]

After exposure to a pure tone, maximum TTS occurs at frequency approximately one-half octave above the exposure tone. If exposure is to octave-band noise the maximum TTS occurs at a frequency one-half octave above the mid-point of the octave band noise.

As information has accumulated regarding the relationship of noise exposure and hearing loss, various proposals have been made regarding how long certain sound pressure

levels may be permitted without causing significant hearing loss. These proposals have ranged from as low as 70 dB to as high as 120 dB.

In 1965 the Committee on Hearing, Bioacoustics and Biomechanics of the National Academy of Sciences and National Research Council (CHABA), an advisory group to the United States Armed Forces and other governmental agencies, proposed certain damage risk critera[3] (FIG.2). It was decided that noise levels were acceptable if a lifetime of daily exposure produced no more than 10 dB of NIPTS at 1000 Hz or below, 15 dB at 2000 Hz, or 20 dB at 3000 Hz and above in the average worker. It is fairly well established that the long-term NIPTS from daily exposure to a given noise will not exceed the TTS produced by a single eight-hour exposure, i.e. TTS_2 (TTS measured two minutes after leaving the noise) will be approximately equal to the average NIPTS occurring after a lifetime of exposure[4]. The equal-energy hypothesis, which assumes that all exposure to a given amount of energy in a given octave band are equally noxious on a time-intensity relationship, was also applied. If 85 dB SPL is tolerable for eight hours then 88 dB is permitted for four hours, 91 dB for two hours, etc.

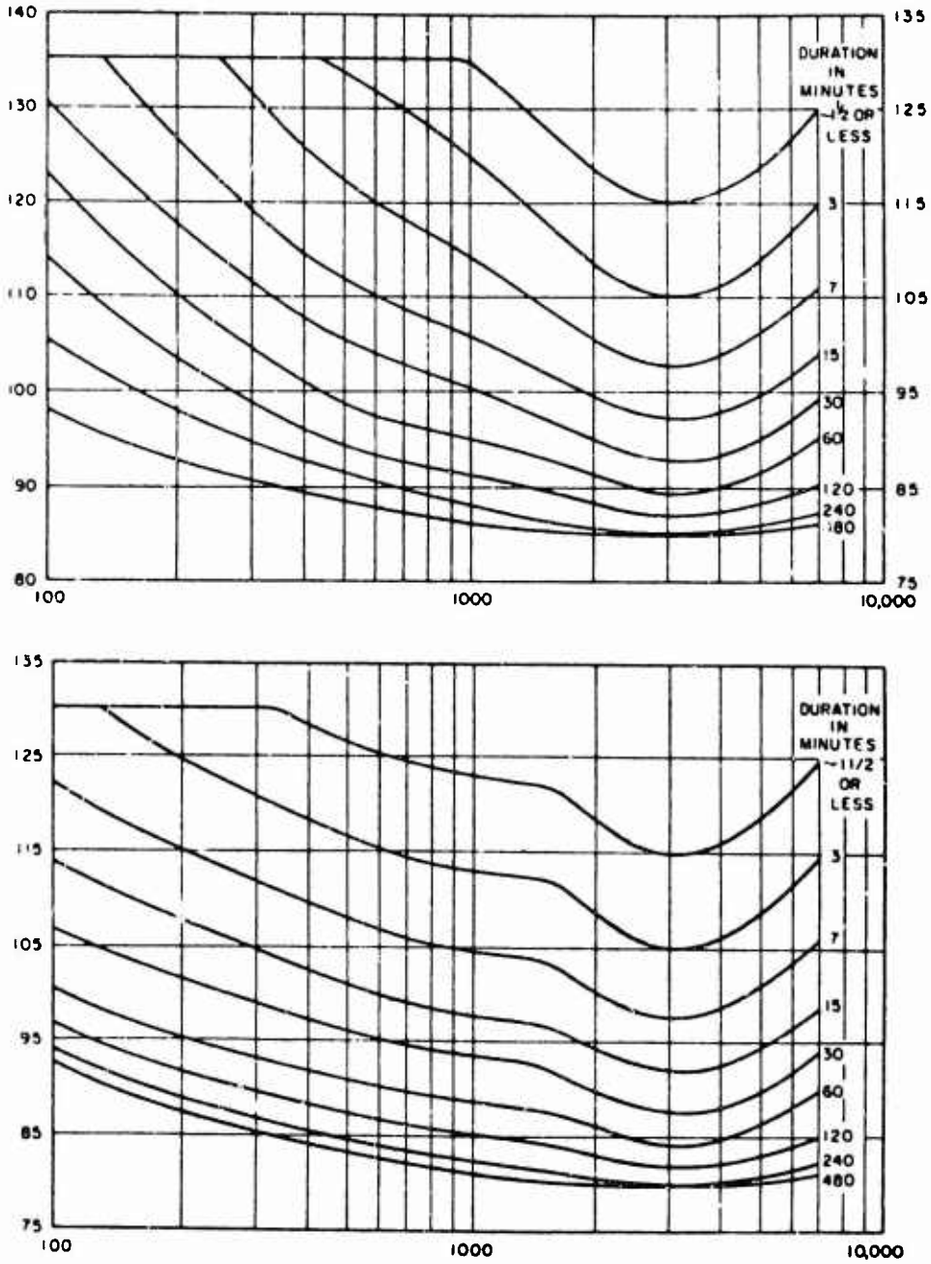

FIG. 2. Upper graph: Damage-risk contours for one exposure per day to full octave (left-hand ordinate) and one-third octave or narrower (right-hand ordinate)bands of noise. This graph can be applied to the individual band levels present in broadband noise. Lower graph: Damage-risk contours for one exposure per day to pure tones.(From Kryter, K.D.et al: Hazardous Exposure to Intermittent and Steady-State Noise. Jour.Acoust.Soc.Amer.,39:451-464,1966. With permission of the authors and the Journal of the Acoustical Society).

Miller[5] has developed curves showing hypothetical growth and recovery of threshold shift. These are shown in FIG. 3 and FIG. 4. The straight dashed line indicates a predicted recovery from a threshold shift induced by exposure to 90 dB for 102 minutes. The large dots represent actual measurements, but Miller points out that while this is probably true for short exposures and small threshold shifts, the theoretical curves are more nearly accurate for TTS induced by exposures of long duration, high intensities, or both, which result in shifts in excess of 35-45 dB. If the exposure were 120 dBA for 7 days (it is very unlikely that a person could tolerate noise of this intensity for 7 days) as shown by the top dashed line, there would be a residual permanent threshold shift of approximately 50 dB from which there would be no recovery.

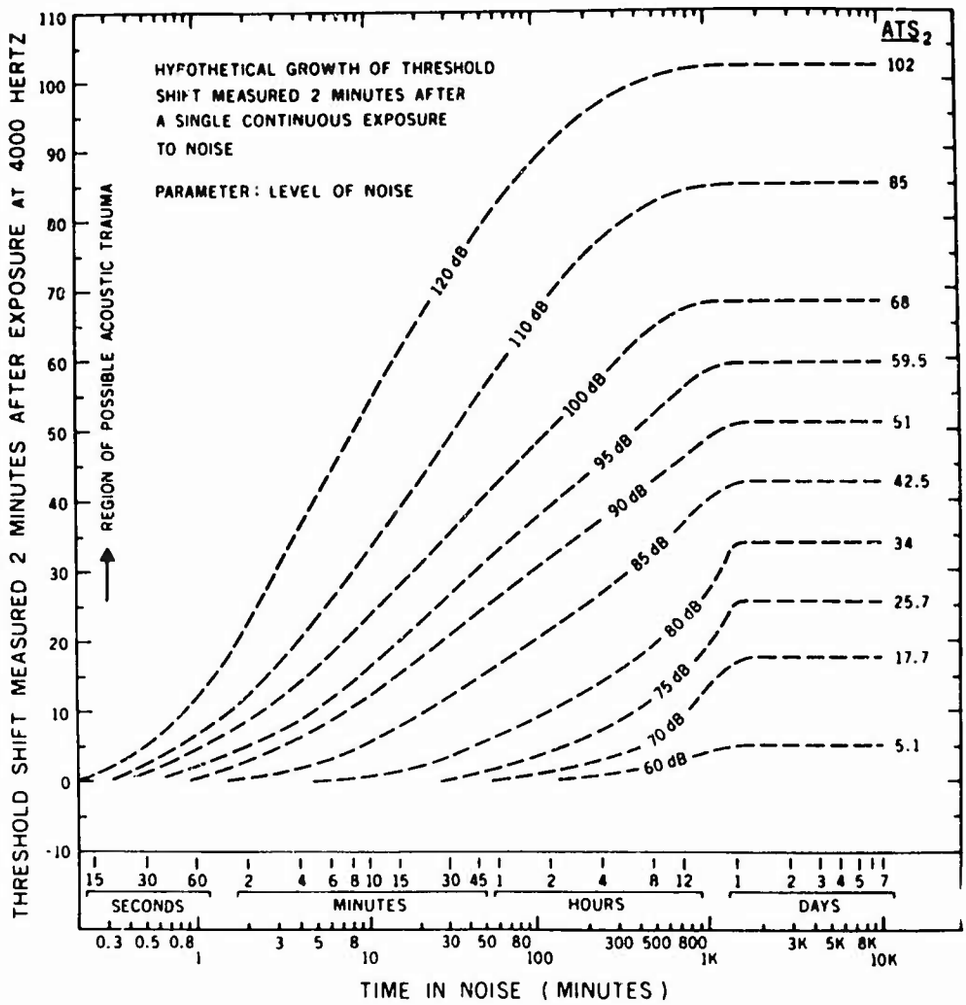

FIG. 3. Hypothetical growth of threshold shift after various single and continuous exposure to noise. (Miller, J.D.: Effects of noise on people. J. Acoust.Soc.Am.,Vol.56, No.3, September, 1974, p.734. With permission of the author and the Journal of the Acoustical Society of America).

The effect of noise-induced hearing loss on the individual is shown in FIG. 5. This is the relation between average hearing threshold level at 500, 1000, and 2000 Hz and degree of handicap as defined by the Committee on Hearing of the American Academy of Ophthalmology and Otolaryngology. It is my personal opinion that these limits are too liberal, and that a person with an average hearing threshold level of 40-55 dB at 500, 1000, and 2000 Hz in the better ear has frequent difficulty with normal speech, requires hearing amplification, and has more than a mild handicap. Miller[5] states, "People with partial deafness from exposure to noise do not live in a world that is simply 'muffled'. Even those sounds that are heard may be distorted in loudness, pitch, apparent location, or clarity. While a hearing aid can be useful to a person with noise-induced hearing loss, the result is not always satisfactory. The modern hearing aid can amplify sound and make it audible, but it cannot correct for the distortions that often accompany injury to the organ of Corti." Anyone who has had to manage a patient with noise-induced hearing loss can appreciate and echo these words.

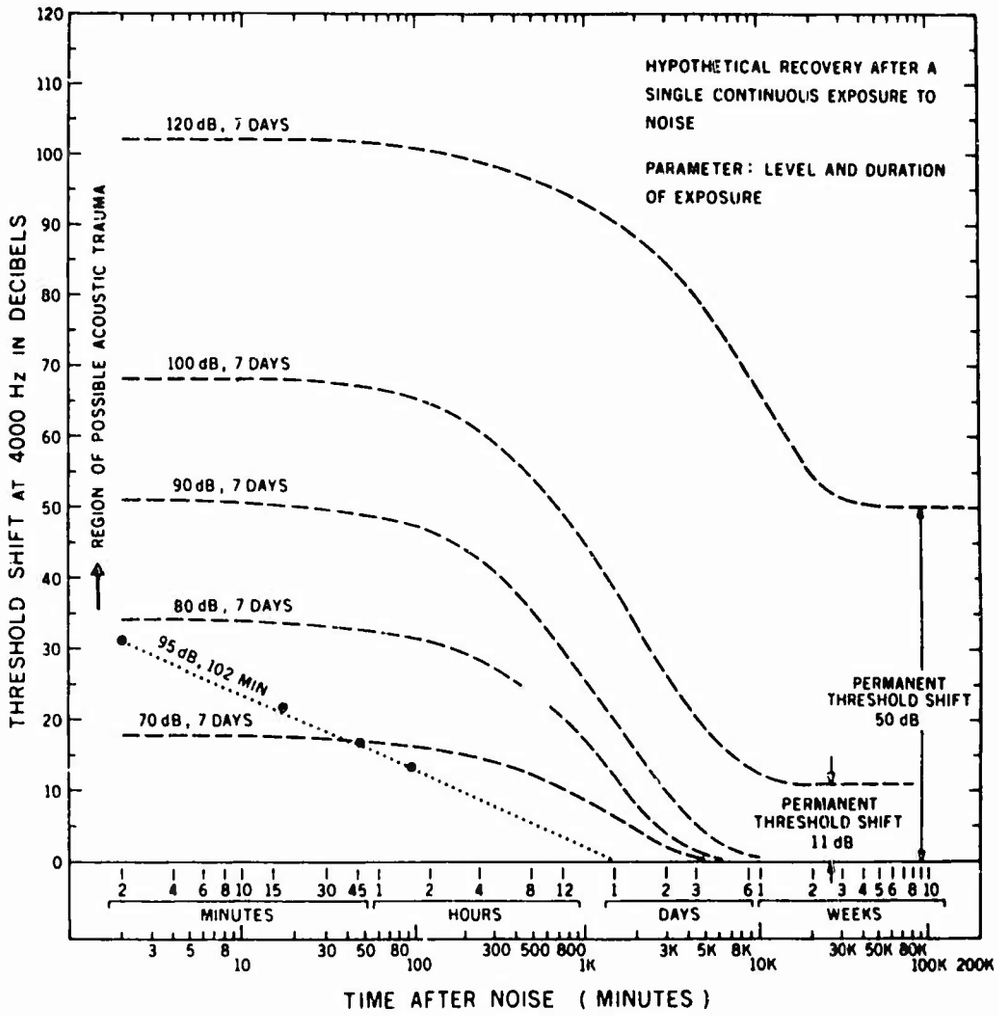

FIG. 4. Hypothetical recovery from threshold shift after various single and continuous exposures to noise. (From Miller, J.D. Effects of noise on people. J.Acoust.Soc.Am.,Vol.56,No.3, September 1974,p.735. With permission of the author and the Journal of the Acoustical Society of America).

ANNOYANCE

Annoyance is a subjective interpretation of the degree of unwantedness of a sound. Kryter calls this perceived noisiness, and attempted to quantify it by subjective judgment tests.

Loudness is that attribute of auditory sensation in terms of which sounds may be ordered on a scale extending from soft to loud and whose unit is the sone. Noisiness, is that attribute of auditory sensation in terms of which noises may be ordered on a scale extending from noticeable to annoying and whose unit is the noy. A sound of 2 noy is subjectively twice as noisy as a sound of 1 noy; 3 noy is three times as noisy as a sound of 1 noy, etc. Perceived Noise Decibels (PNdB) is the unit of perceived noise level (PNL) and is the translation of the subjective noy scale to a dB-like scale.

Equal noisiness contours can be obtained by having subjects adjust octave band levels in broadband, random noise until the noise is perceived as equally noisy to a standard band centered at 1000 Hz. Equally noisy in this case means one sound would not be preferred over the other in their home, day or night. Kryter modified this to account for frequency components of tones (Tones Corrected Perceived Noise Level) (PNLT) and the duration of the noise signal since long duration noise exposures are usually more annoying than short duration exposures. This was labelled the Effective Perceived Noise Level (EPNL). These terms have been applied most extensively in the field of aircraft noise. The Federal Aviation Administration uses these terms in much of their measurements, and it exceeds the A-weighted noise levels by 11-17 dB depending on corrections. Since the PNLT measurements require complex instrumentation and analysis they have not been widely applied.

CLASS	DEGREE OF HANDICAP	AVERAGE HEARING THRESHOLD LEVEL FOR 500,1000 and 2000 IN THE BETTER EAR		ABILITY TO UNDERSTAND SPEECH
		MORE THAN	NOT MORE THAN	
A	NOT SIGNIFICANT		25 dB	No significant difficulty with faint speech
B	SLIGHT HANDICAP	25 dB	40 dB	Difficulty only with faint speech
C	MILD HANDICAP	40 dB	55 dB	Frequent difficulty with normal speech
D	MARKED HANDICAP	55 dB	70 dB	Frequent difficulty with loud speech
E	SEVERE HANDICAP	70 dB	90 dB	Can understand only shouted or amplified speech
F	EXTREME HANDICAP	90 dB		Usually cannot understand even amplified speech

FIG. 5. Guideline for the relations between the average hearing threshold level for 500, 1000, and 2000 Hz and degree of handicap as defined by the Committee on Hearing of the American Academy of Ophthalmology and Otolaryngology. (From Davis, H.: Classification-Evaluation of Hearing Handicap. Trans.AAOO 69, July-Aug.1965, p.741. With permission of the author and the Transactions of the American Academy of Ophthalmology and Otolaryngology).

Most investigators continue to use the A-weighted sound level meter and record noise levels for various periods of time. Measures of annoyance are then related to these time-noise relationships. Some of the commonly used measures are:

1. Energy Mean Noise Level (L_{eq})[8], or equivalent continuous noise level, is an A-weighted measure which accounts for duration and magnitude of all sounds occurring during a given period.

2. Composite Noise Rating (CNR)[9,10], is generally used in the United States for the evaluation of noise environment. It is based on the concept that behavioral response of people to noise at a particular location is a function of the sum of the perceived noise levels occurring regularly during a 24 hour period with a 10 dB difference at night-time due to increased sensitivity by people from sleep interference.

3. Noise Exposure Forecase (NEF)[11] is similar to CNR, but in addition it accounts for duration and pure tone content of each single noise event.

4. Day/Night Average Sound Level (L_{dn})[12] the average, A-weighted sound level over a 24 hour period, with a 10 dB penalty between 10:00 a.m. and 7:00 a.m.

5. Community Noise Equivalent Level (CNEL)[13] has been adopted by California and is essentially an L_{eq} for 24 hours with a 5 dB penalty from 7:00 p.m. to 10:00 p.m. and a 10 dB penalty from 10:00 p.m. to 7:00 a.m.

6. Noise and Number Index (NNI)[14] is a measure based on Perceived Noise Level with weighting factors added to account for the number of noise events, and used in some European countries for rating the noise environments near airports.

7. Noise Pollution Level (L_{NP} or NPL)[15] is a measure of the total community noise applicable to both traffic and aircraft noise. It is based on the average sound level (L_{eq}) and the magnitude of the time-varying noise level.

These unfortunately are not the only terms used but they are presented to provide the reader with some of the terms employed to describe noise exposures.

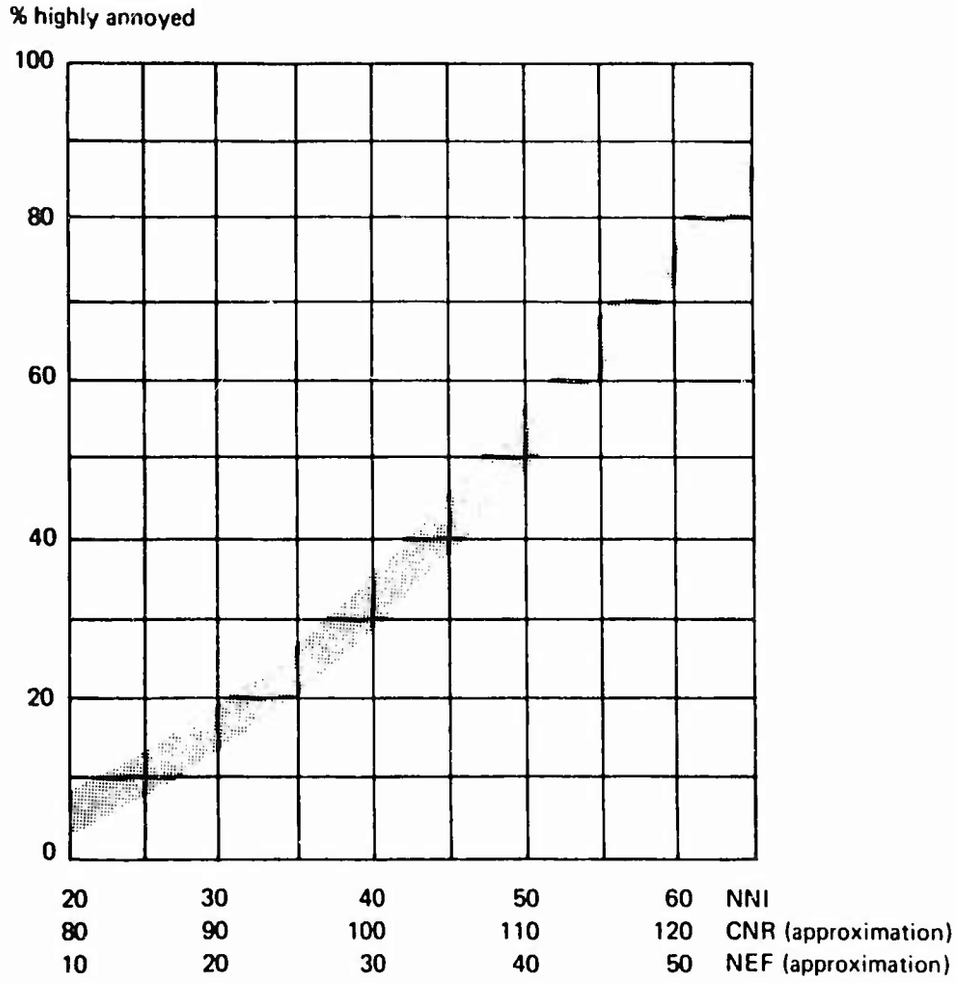

FIG. 6. Percentage of Highly Annoyed Persons in Relation to Noise. (From Public Health and Welfare Criteria for Noise, July 27, 1973, U.S.Environmental Protection Agency, Washington, D.C. 20460, p.3-3).

ANNOYANCE AND COMMUNITY RESPONSE

The degree of annoyance and the community response to noise can be correlated. Individual factors related to the annoyance of a noise include,

1. The intensity level and spectral characteristics of the noise.
2. The duration of the noise event.
3. The presence of discrete frequency components.
4. The presence of impulses.
5. The abruptness of onset or cessation of the noise event.
6. Degree of hoarseness or roughness of the noise.
7. Degree of intermittency in loudness, pitch, or rhythm.
8. The information content.
9. The degree of interference with activity.[8]

This response is variable and includes:

1. The background noise against which a particular noise event, such as aircraft flying occurs.
2. The previous experience of the community with the particular noise.
3. The time of day during which the intruding noise occurs.
4. Attitude of people toward the noise makers and the contribution of the activities associated with the noise source to the general well-being of the community.
5. Socio-economic status and educational level of the community.
6. Fear associated with activities of noise sources such as fear of crashes in the case of aircraft noise.[8]

FIG. 6 shows the percentage of persons highly annoyed by noise as determined by several rating systems. The significant point to be made here is that no matter how you measure it or what scale is used, when the energy and aversive context of a noise reaches a certain level, people will start to complain.

Table 1. shows the percentages of persons who are highly annoyed and also register complaints. It is estimated that only 20% of those annoyed actually register a complaint.[16]

L_{dn}	PERCENTAGE OF HIGHLY ANNOYED	PERCENTAGE OF COMPLAINTS
50	13	Less than 1
55	17	1
60	23	2
65	33	5
70	44	10
75	54	15
80	62	Over 20

TABLE 1. Percentages of Persons Highly Annoyed who Register Complaints as a Function of L_{dn}. (From Public Health and Welfare Criteria for Noise, July 27, 1973, U. S. Environmental Protection Agency, Washington, D.C. 20460, p.3-5).

SLEEP INTERFERENCE

Studies have shown that sleep is not one continuous pattern but occurs in various stages. They are categorized 1, 2, 3, 4 and REM (for rapid eye movements) based on various EEG patterns which occur during the different stages of sleep[17].

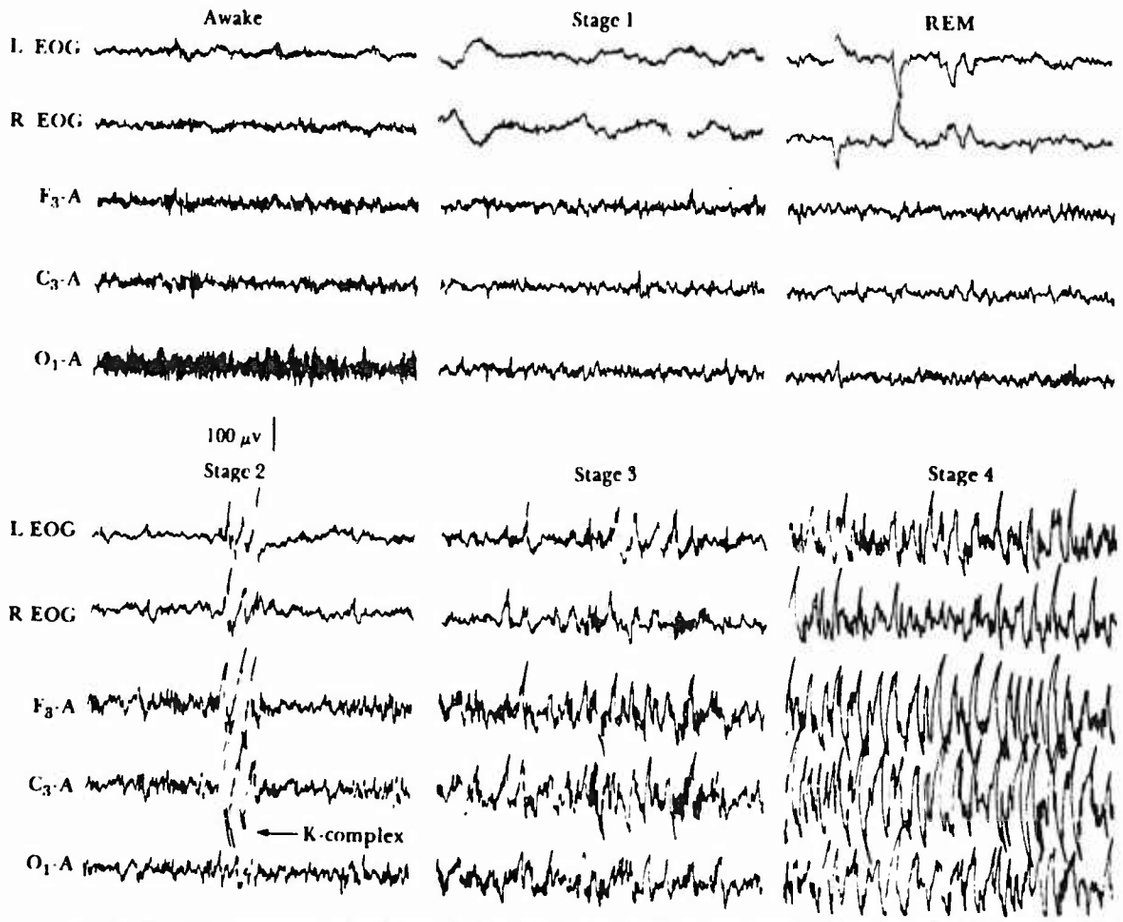

FIG. 7. EEG stages of sleep following revised scoring criteria of Rechtschaffen and Kales. The bursts of alpha are clearly seen in the O_1-A lead during REM sleep. A K-complex is seen in the middle of Stage 2. (From Johnson, L.C.: Are Stages of Sleep Related to Waking Behavior? American Scientist, Vol. 61, No.3, May-June, 1973, pp.326-338. With permission of the author and the American Scientist).

FIG. 7 shows these various EEG patterns. From a waking state with high-amplitude 9-11 Hz alpha activity, a person enters Stage 1 which consists of a low-amplitude mixed-frequency EEG activity without sleep spindles, K-complexes, or rapid eye movements (REM). In Stage 2 there is a low-amplitude mixed frequency activity with K-complexes and 12-14 Hz sigma rhythms (sleep spindles). In Stage 3, twenty percent of the EEG is high amplitude

slow delta waves and by Stage 4, fifty percent of the tracing shows these delta waves. During REM sleep there is low amplitude mixed-frequency EEG activity similar to Stage 1 but with bursts of REMs and a marked decrease in tonus of certain head and neck muscles.

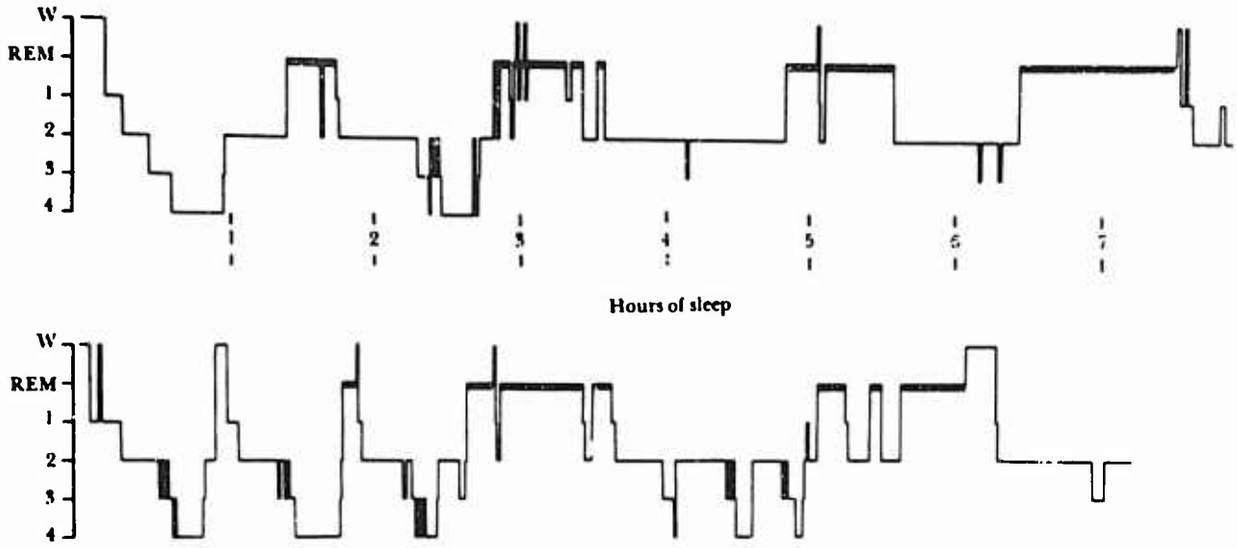

FIG. 8. All-night sleep profiles, illustrating sleep cycles, for two young adult male subjects. (From Johnson, L.C.: Are Stages of Sleep Related to Waking Behavior? American Scientist, Vol. 61, No.3, May-June 1973, pp.326-338. With permission of the author and American Scientist).

FIG. 8 shows a typical sleep profile for two young adults. As can be seen, from waking, one enters Stage 1, remains for a while, then enters Stage 2, and so on until Stage 4 is reached. Then the sleep pattern jumps back to Stage 2, and after a while, enters REM sleep without going through Stage 1. This usually occurs about 90-100 minutes after sleep onset. A person then returns to Stage 2, possibly Stage 3 or 4 then back to REM. Note also that several waking periods occur during each sleep period. The periods of REM sleep continue to occur at 90-100 minute intervals all night with REM sleep growing longer as hours of sleep progress while Stages 3 and 4 decrease. These patterns occur regardless of what time of day sleep onset occurs.

From adolescence to age forty, one spends approximately 6% sleep time in Stage 1, 50% in Stage 2, 7% in Stage 3, 16% in Stage 4, 20% in Stage REM, and about 1% of sleep is occupied by body movements. With increasing age, Stage 3 and 4 sleep decreases, wakings increase but REM sleep remains fairly constant. Stage 4 is completely absent in elderly persons.

REM sleep, when most dreaming occurs, and Stage 4 sleep have received considerable attention since deprivation of either of these stages results in a rebound phenomenon with earlier and more frequent attempts to enter these stages during recovery sleep. Researchers concluded that there must be a physiological need for both REM and slow-wave sleep. Dement[17] reported in 1960 that dream deprivation (REM deprivation) resulted in increased anxiety, irritability and difficulty in concentration. Subsequent investigators could not confirm the findings, and by 1965 Dement[18] stated he no longer believed REM sleep occurred to satisfy a need for dreaming. Johnson[19] deprived subjects of REM and Stage 4 sleep, then tested performance and psychological function. He found no decrement in mental r motor performance. It is known that man cannot function effectively without sleep, but no evidence is available to show that any particular stage of sleep is required to perform adequately.

Regardless of whether noise is more deleterious at one stage of sleep than at another the fact remains that noise can awaken a sleeping person, and that sleep deprivation adversely affects psychological well-being and motor performance.

FIG. 9 is a composite of studies showing wakings to sounds from various laboratories. It is clear that noise louder than 35 dBA can cause arousals in young adults. Arousals occur more frequently with increasing intensity.

Many factors influence arousal from sleep. These include: (a) motivation to awake; (b) sex (women awake more easily than men); (c) age (older awake more easily); (d) sleep deprivation (more easily awakened if well rested); (e) time in sleep cycle (more easily awakened later in sleep period); (f) stimulus meaning and familiarity; and (g) adaptation to noise.

A recent study[20] on the effects of intermittent noise in the form of high-pitched tonal pulses every 22 seconds, 24 hours per day presented at 80, 85 and 90 dBA for 10 days each, showed no increase in time to reach early Stage 2, slight decrease in Stage 4 but no decrease in Stage REM. There was a significant change in heart rate, finger pulse

amplitude and EEG evoked response during sleep, which was not observed during the day[21].

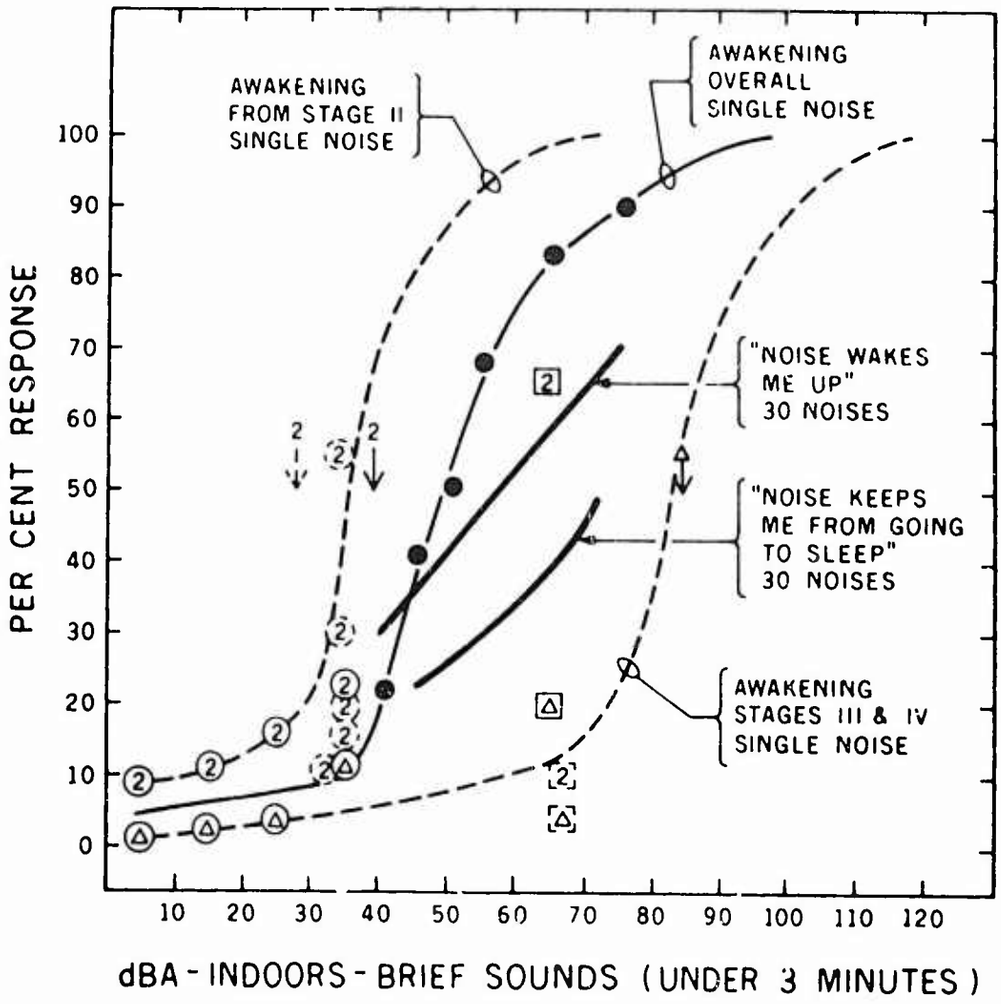

FIG. 9. Wakings to sound from various laboratory and questionnaire studies are shown. The horizontal axis gives the approximate A-weighted sound level (dBA) of the noise. (From Miller, J.D.: Effects of noise on people. J.Acoust.Soc.Am., Vol.56,No.3, September 1974. With permission of the author and Journal of the Acoustical Society of America).

SPEECH INTERFERENCE

FIG. 10 is a recent offering by Webster[22] of the effect of noise on speech communication. Three main factors are involved: background noise, distance from speaker to listener, and volume of the voice. Secondary considerations are the degree of articulation, age of speaker and listener (children hear better but speak with less precision; the reverse is usually true for older age), and visual cues. Webster has very nicely compiled the various speech interference effects of noise. This one chart includes subjective evaluations of the noise, the distance-noise areas with a correction for expected voice levels and communicating voice levels, and finally a conversion table accounting for low-frequency noise. This allows anyone with a sound level meter and a little training to make fairly complex measurements. The Speech Interference Level Four (SIL-4) is the average of four octaves centered at 500, 1000, 2000 and 4000 Hz. The C-weighting scale is a nearly flat scale and gives increased weight to low frequency noise which is commonly a source of speech interference. The C scale matches the response of the ear to high intensity noise. The A-weighted scale discriminates against low frequency sounds and supposedly matches the response of the ear to low intensity noise. Using the contours included by Webster, the SIL-4 can be calculated by measuring the sound on the C scale, then the A scale, compute the difference, and starting from the A-weighted level, read up until the contour crosses the line (C-A), then read vertically to the SIL-4.

Another speech interference problem is laryngeal strain caused by trying to speak in a noisy area. Workers in noisy areas usually learn to substitute visual for auditory signals or to go very close to speak to a fellow worker. Occasionally, however, cases of chronic vocal strain from speaking in noise are seen.

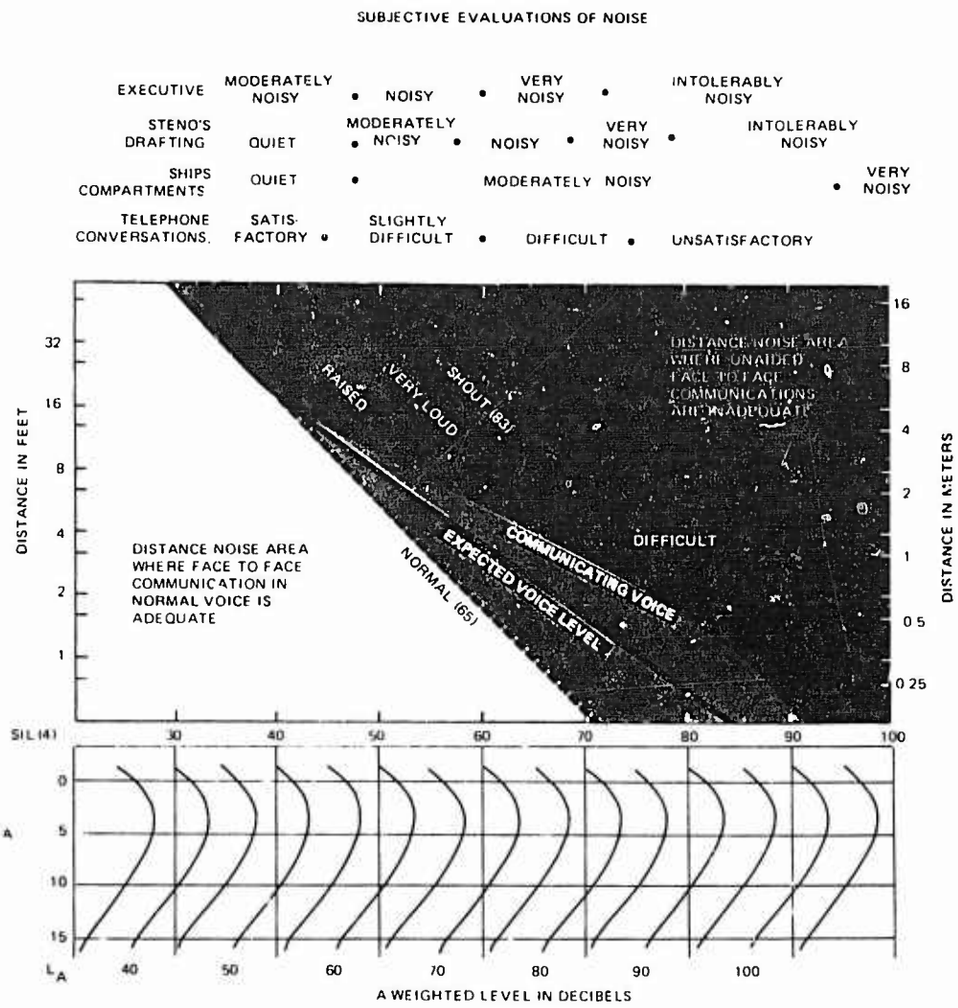

FIG. 10. Permissible distance between a speaker and listeners for specified voice levels and ambient noise levels. Webster, J.C. and Cluff, G.L.: Speech Interference by Noise. Inter-Noise 74 Proceedings of the International Conference on Noise Control Engineering. ed. J.C.Snowdon. State College, Pa.ARL-PSU 1974, pp.553-558. (With permission of the authors and INCE).

PSYCHOLOGICAL AND SOCIOLOGICAL EFFECTS

Mental and motor performance in work has been studied. Broadbent[23] found that noise must reach approximately 90 dB before it affects non-auditory work performance, with frequencies above 2000 Hz having more effect than lower frequencies.

Broadbent[23] and Shambaugh[24] further showed that, in general, subjects having personality traits of "anxious", "introv rted", and "somatic responsive" were more adversely affected by noise on mental tests (I.Q. and arithmetic) and motor tasks (reaction time and tracking); however, much of the work in this field is not conclusive, and although there is data to support somewhat poorer performance on tests, there is conflicting data depending on what performance scores are averaged.

Helper[25] studied the performance on a complex counting task and measured three physiological variables (skin conductance, pulse interval and muscle tension), on 24 subjects in quiet for one hour, and again when exposed to 110 dB noise for one hour. He found no difference between the performance scores but did repor' an increased skin conductance.

Blackwell and Belt[26] found no significant difference in performance of vigilance tests when subjects worked in 50 dB, 75 dB and 90 dB ambient noise levels.

Hershman and Lowe[27] found that prolonged exposure to intermittent noise in the 3000-4000 Hz range at 80, 85, and 90 dBA for 10 days each had no adverse effects on mental or motor performance.

Abey-Wickrome et al[28] reported a higher incidence of admissions to mental hospitals from areas with high levels of aircraft noise. These authors concluded that noise did not actually cause mental illness, but was one additional factor in increasing the admissions to psychiatric wards.

Jansen[29], Andriukin[30] and Shatalov[31], among others, have reported an increased incidence of digestive, metabolic, neurological and psychiatric problems among workers in industries with high noise levels.

These studies are by no means conclusive. No adverse psychological effects were noted by Seymour[32] in his study of the effects of intermittent noise exposure 24 hours daily for 30 days. More work on the effects of noise on psychological health are indicated.

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PHYSIOLOGICAL EFFECTS OF NOISE

Robert W. Cantrell
Captain, Medical Corps, United States Navy
Chairman, Department of Otolaryngology
Naval Regional Medical Center
San Diego, California 92134

SUMMARY

It is well known that noise adversely affects mankind. Many studies have been performed which show the effects of noise on hearing, speech reception, sleep, mental health and work performance. Until recently, relatively little had been written on the physiological effects of noise. Studies which have been reported are generally retrospective on a group of people working in a noisy environment where precise controls over the intensity of the noise and the duration of exposure were lacking. Recent studies show that the effect of noise on the biochemistry of the body, the cardiovascular system, and the organ systems controlled by the autonomic nervous system are more serious than previously suspected.

Noise serves as a stressful stimulus which provokes the General Adaption Syndrome as described by Selye. Noise is one of the several stressful stimuli which activate this syndrome via the hypothalmus to the pituitary which produces ACTH resulting in increased adrenocortical activity. There is considerable evidence to support this concept, and this theory along with recently carefully controlled studies, are reviewed.

INTRODUCTION

Noise has been recognized as harmful to man for at least 2500 years. The Sybarites of Greece banned metalwork involving hammering within the city limits as early as 600 B.C.[1] As industrialization has increased along with population density, noise levels have risen to a point where they have become a serious public health problem.

The effects of noise on hearing, annoyance, sleep and speech interference, and work performance have been studied extensively. Until recently relatively little has been written on the physiological effects of noise. Most of the work has been done as retrospective analysis on groups of people working in a noisy environment where precise measures of the intensity of the noise and the duration of exposure were lacking. In the studies, it was not always possible to separate the effects of heat, light, dust, noxious fumes or other environmental pollutants from the effects of noise alone.

Recent studies show that the effect of noise on the biochemistry of the body, the cardiovascular system, and the organ systems controlled by the autonomic nervous system are more serious than previously suspected.

In Germany, Jansen[2], Lehmann[3], and Meyer-Delius[4]; in Russia, Andriukin[5], Andrukovitch[6] and Shatalov[7] among others; and in the U.S. Davis[8], Rosen[9] and more recently Cantrell[10], have studied the physiological effects of noise exposure. A symposium held in Boston in 1969 was devoted to this subject, and resulted in a book[11] which detailed much of the knowledge available at that time.

PATHWAYS

A pathway from the sound source to the target organ must be established in order to show a cause-and-effect relationship. For the purposes of this discussion, infra-sound (below 20 Hz) and ultra-sound (above 20,000 Hz) will not be considered. Nor will the possible physiological effects of vibratory energy on the body in general be considered. There is evidence that vibratory energy can affect the body; that the vibrations transmitted through structures is more significant than airborne transmissions; and that vibrations (sound) from 20 to 20,000 Hz may exert a physiological effect in ways other than through the auditory mechanism, i.e.through the skin. These studies are scarce and since the most damaging airborne vibratory (noise) effects are transmitted through the ear with its central auditory connections, it is those physiological effects which will be considered here.

Once sound enters the auditory canal, it causes the tympanic membrane to vibrate. This in turn moves the three ossicles which at the oval window create a wave in the inner ear fluids by the piston-like action of the footplate of the stapes. The relatively large area of the tympanic membrane compared with the small area of the stapedial footplate (17:1)plus a small lever advantage from the ossicles, transforms the small pressure of sound energy impinging on the tympanic membrane into a 22-fold greater force acting on the inner ear fluid. The fluid wave thus created distorts the basilar membrane and the hair cells of the organ of Corti are stimulated. Nervous impulses generated in the organ of Corti travel along the auditory neurons to the central auditory nuclei.

Sound is also transmitted through the bones of the skull directly to the inner ear, and we measure these two pathways (air conducted and bone conducted sound) to help diagnose hearing disorders. One who cannot hear air conducted sound but can hear by

bone conduction has a conductive hearing loss which in most cases can be remedied by appropriate medical or surgical treatment. If one has difficulty hearing both air and bone conducted sound, this is known as nerve deafness which is not correctable, and sound must be amplified in order for the patient to hear.

After stimulating the auditory nerve, the sound waves, which are now nervous impulses, travel to the central auditory nuclei in the medulla, where some fibers ascend through the midbrain via the lateral lemnisci on the same side, but more cross before ascending on the opposite side through the midbrain to reach the inferior colliculus, then the medial geniculate body and finally the auditory area of the temporal lobe where the sound is interpreted.

It is probable that after reaching the central auditory nuclei, impulses travel through the reticular formation to reach the hypothalamic nuclei. From the hypothalamic nuclei which are situated just superior to the pituitary, the products of stimulation travel to the pituitary which then produces endocrine effects and completes the auditory-hypothalamic-pituitary-endocrine pathway.

The hypothalamus is not the only part of the brain directing autonomic activity. The forebrain, the thalamus and the cerebral cortex are all integrated with the hypothalamus to utilize behavioral and autonomic adjustments which serve to adapt the individual to changes in both the internal and external environment.

AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system (ANS), also known as the vegetative nervous system, is a system of motor neurons whose cell bodies are collected into ganglionated chains in the thoracic region near the vertebral column and in isolated ganglions elsewhere in the body. Anatomically the A.N.S. is divided into the thoracolumbar (sympathetic) and craniosacral (parasympathetic) division. This system is generally not under voluntary control.

Table I lists the functions of the autonomic nervous system, which acts to maintain the constancy (homeostasis) of the fluid environment (internal milieu) of the body. The autonomic nervous system combats forces which tend to cause variations in this environment. It regulates the composition of body fluids, their temperature, quantity and distribution by effecting changes in circulatory, respiratory, excretory and glandular organs.

TABLE I

AUTONOMIC NERVOUS SYSTEM FUNCTION

SYSTEM	PARASYMPATHETIC (CHOLINERGIC)	SYMPATHETIC (ADRENERGIC)
Eye (Pupil)	Constricted	Dilated
Heart Rate	Decreased	Increased
Blood Vessels		
Coronary	Dilated	Dilated
Skin & Mucosa	Dilated	Constricted;Dilated
Skeletal Muscle	Dilated	Dilated;Constricted
Cerebral	Dilated	Constricted (?)
Pulmonary	Dilated	Constricted
Abdominal Viscera	Dilated	Constricted
Bronchi	Constricted	Dilated
Glands		
Sweat	Stimulated	Stimulated
Salivary	Stimulated (Thick)	Stimulated (Watery)
Gastric	Stimulated	Inhibited
Adrenal		Stimulated
Smooth Muscle		
Skin (Pilomotor)		Contracted
Stomach	Increased Tone	Decreased Tone
Small & Large Intestine	Increased Tone	Decreased Tone
Bladder	Contraction	Inhibition
General	Restorative	Energizing
Mediator	Acetylcholine	Sympathin Epinephrine Norepinephrine

The two divisions are antagonistic: one slows, the other speeds the cardiac rate;one constricts, the other dilates the pupil or the bronchi. Generally, the sympathetic strengthens the defense against such challenges as enemy attacks, temperature variations, and water deprivation. Animals who have had sympathectomies are incapable of working (no sugar is mobilized); cannot withstand exposure to temperature extremes (no sweating when hot; no vasoconstriction when cold) and they are less able than normals to withstand oxygen deprivation or hemorrhage. They can survive in a controlled environment.

The parasympathetic system is concerned with restoration of the body processes. It inhibits the heart rate, contracts the pupils and stimulates the digestive tract through which the energy stores of the body are replenished. It is primarily in control while one is sleeping.

DISCUSSION

Selye[12] described the General Adaptation Syndrome. According to this concept, an individual exposed to stress - cold, heat, drug reaction, fractures, infections, operations, burns or other trauma (NOISE) - responds by:

1. Stimulation of the hypothalamus which
2. Stimulates the anterior pituitary to release ACTH which
3. Stimulates the adrenal cortex to release cortisol which
4. Stimulates the body to protect against systemic anabolism of tissue.

This theory is well accepted even if there is not complete agreement as to the actual mechanism of action. Stress is known to be a factor in the development of such diseases as peptic ulcers, cardiovascular disease, including hypertension and coronary artery disease, and it is implicated in the aging process.

Noise, especially aversive, intrusive noise, is thought to be merely one of many agents which serves as a stress-provoking stimulus. Noise stimulates the sympathetic portion of the A.N.S. As such it should be minimized just as noxious fumes, excessive heat or cold or, indeed, even marked population density, should be diminished and controlled where possible in the environment.

Assuming this to be true, what evidence do we have that noise has any effect on these functions?

Mason[13] reported the electrical stimulation of the hypothalamus of conscious Rhesus monkeys was associated with an increase in pituitary-adreno-cortical activity, as judged by the marked elevation of plasma 17-hydroxycorticosteroid.

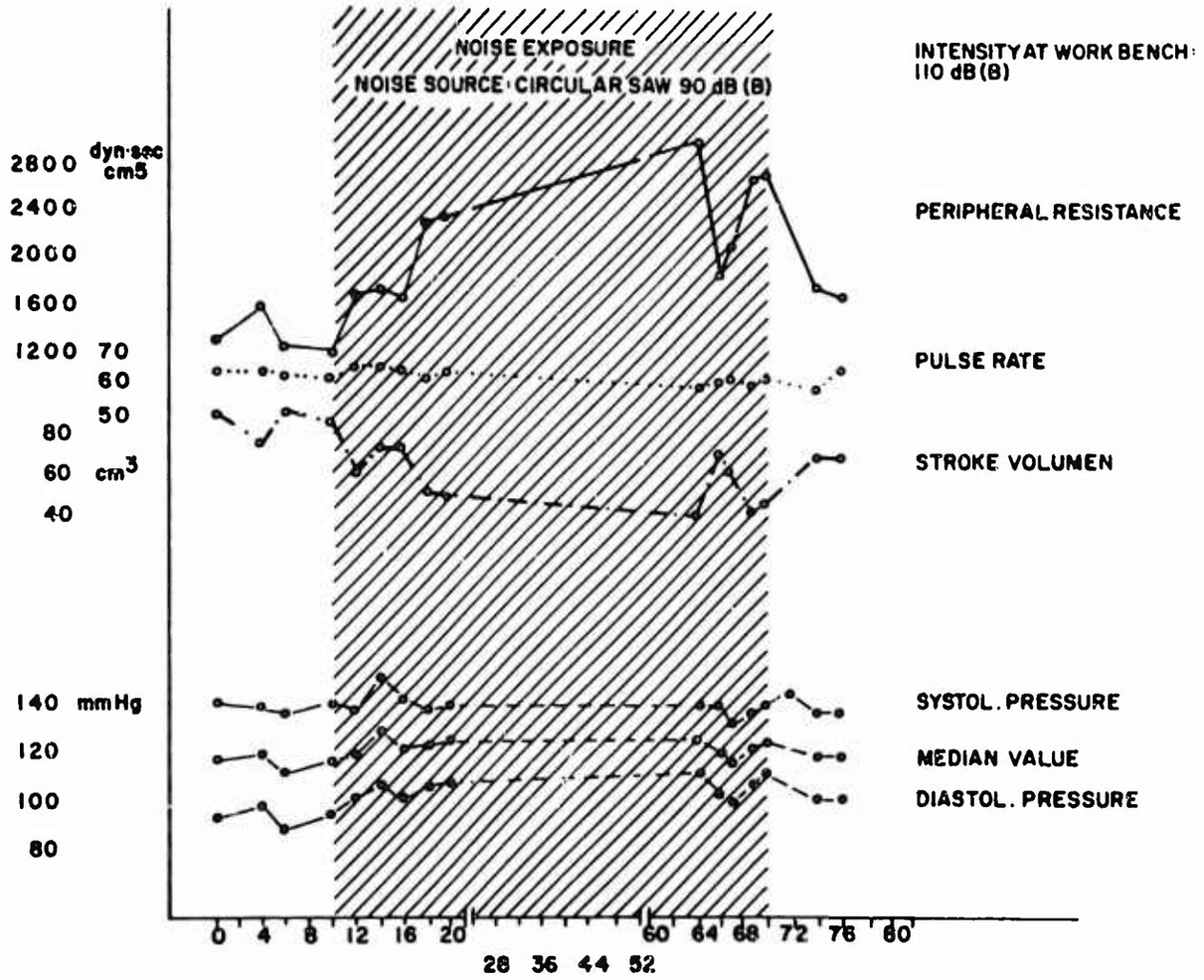

FIG. 1. Circulatory reaction during noise exposure in one subject employed in a noisy factory. (From Lehmann and Tamm. Forsch.-Ber.Wirsch.-u Verkehrsmin, NRW 517 [Kohn-Oplader, 1958], Cited by G. Jansen, Transl.Bel.Inst.Hear.Res. No.26,1972. With permission).

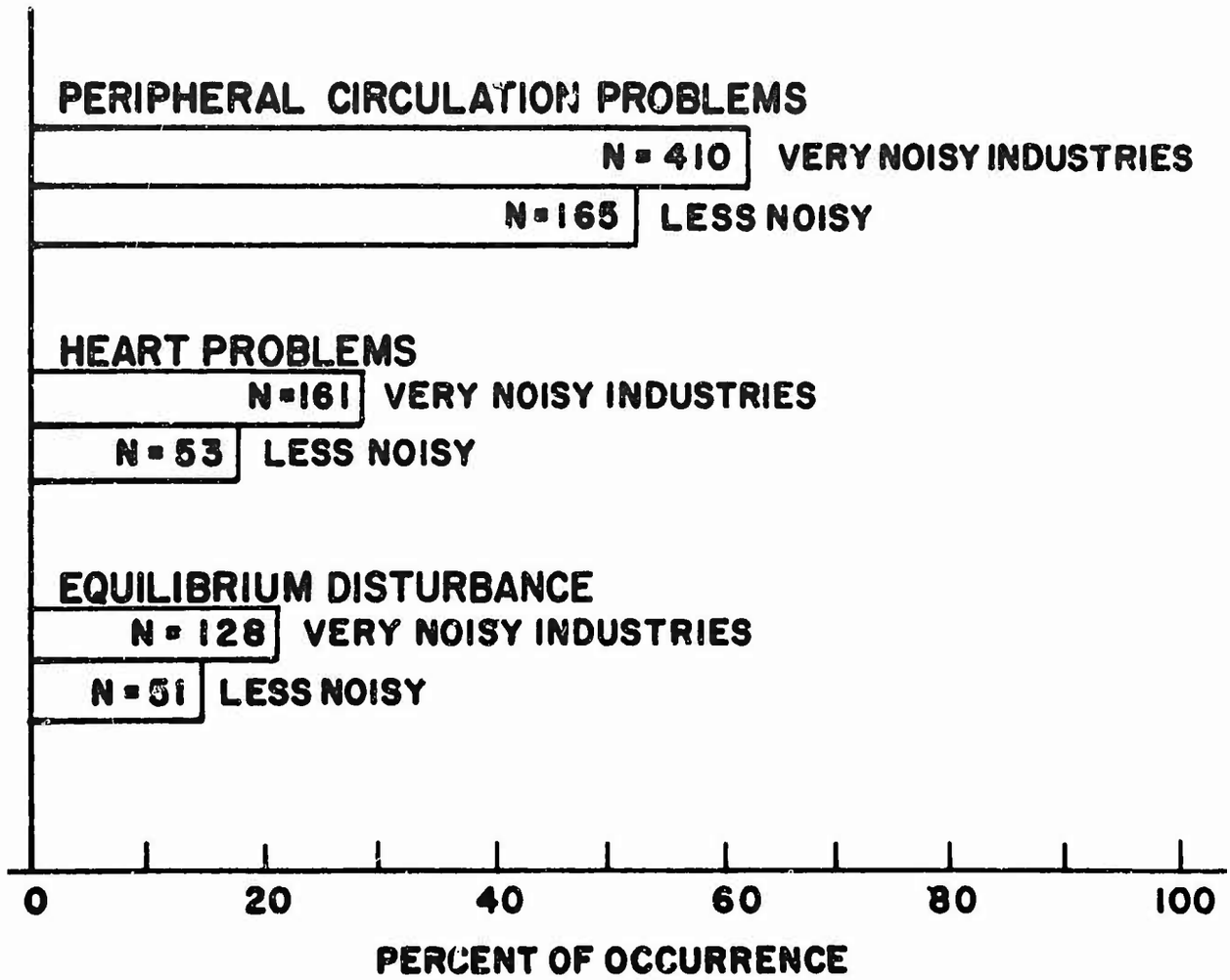

FIG. 2. Differences in percentages of occurrence of physiological problems in 1,005 German industrial workers. The differences in peripheral circulation and heart problems in the two classes of industry were statistically significant. (From G. Jansen, Stahl Eisen,81,1961, pp.217-220. With permission of the author and Stahl.Eisen.).

Henkin and Knigge[14] exposed rats to 130 dB at 220 Hz for 48 hours and measured adrenal secretion of corticosterone. It was found that the output of adrenal corticosterone doubled in 30 minutes and trebled in 60 minutes. This trebled excretion rate was maintained for approximately 12 hours at which time it fell to normal or subnormal levels only to rise again to the former high rate where it was maintained for 25 to 48 hours.

Friedman, et al[15], exposed rabbits to 102 dB of white noise for 10 weeks and showed a much higher level of blood cholesterol than non-exposed animals despite being on identical diets. The animals exposed to noise developed more aortic atherosclerosis and more cholesterol deposits in the iris than the control animals.

Geber[16] exposed gravid female rats to noise intensities of 74-94 dB from 20 to 2,500 Hz for 6 minutes of each hour followed by 54 minutes of quiet (ambient noise level was 64 dB) and to a flashing light for the same period of time, throughout each day of pregnancy or to some other desired day (i.e. 16-20 days).

He found:

1. Total litter resorption occurred in 40-50 percent of the pregnancies.

2. Increased congenital anomalies, including meningoceles, spinal bifida, cranial hematomas, abdominal hernias, and defects of the eye, tail, hind- and forefoot.

Geber and Anderson[17] studied the effects of chronic intermittent noise stress on the body weight and the weight of the ventricles, adrenal glands, kidneys, and ovaries of young and old rats and rabbits. Significantly hypertrophied ventricles of both species were found at the end of three weeks' stress. Body and other organ weights were slightly decreased, with the exception of the adrenals and kidneys of the older rats which were increased.

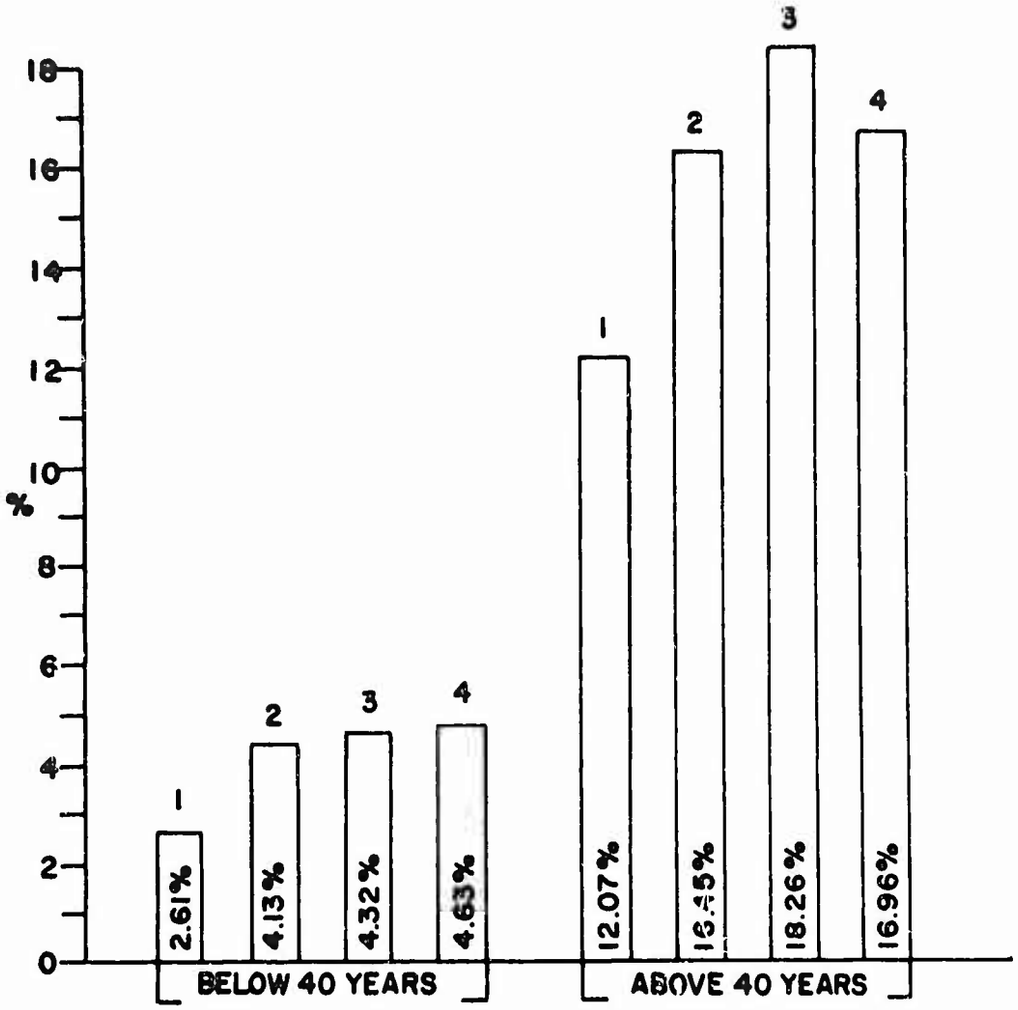

FIG. 3. Incidence of hypertension in male and female workers (in age groups under and above 40 years) in noisy workshops: 1. tool making workshop; 2. sorting workshop; 3. workshop with automatic lathes; and 4. workshop producing ball bearings. (From A.A.Andriukin, Cor.Vassa., 1961, pp.285-293. With permission).

Similar evidence is available in humans. Davis, et al[18] labeled the following set of responses to noise the N-responses:

1. A vascular response characterized by peripheral vasoconstriction, minor changes in heart rate, and increased cerebral blood flow since cerebral vessels show no vasoconstriction to such stimuli.

2. Slow, deep breathing.

3. A change in the resistance of the skin to electricity (Galvanic skin Response [G.S.R.]).

4. A brief change in skeletal muscle tension.

To this set of responses can be added:

1. Changes in gastrointestinal motility.

2. Chemical changes in blood and urine from endocrine glandular stimulation.

A tone of approximately 70 dB SPL at 1,000 Hz will elicit the N-response. This same tone continued for a long enough time may induce TTS or NIPTS and is near the level at which broadband noise may become significantly adverse to people[19].

Davis and Berry[20] and Stern[21] found that humans who could avoid an 80 dB, 10-second 800 Hz tone by pushing a switch at the correct time, exhibited greater gastro-intestinal motility during the tone (i.e. when they failed to press the switch) than did subjects who had no means of avoiding the tones. Kryter[19] labeled this a response-contingent effect of noise. The noise thus became an aversive stimulus, primarily because it

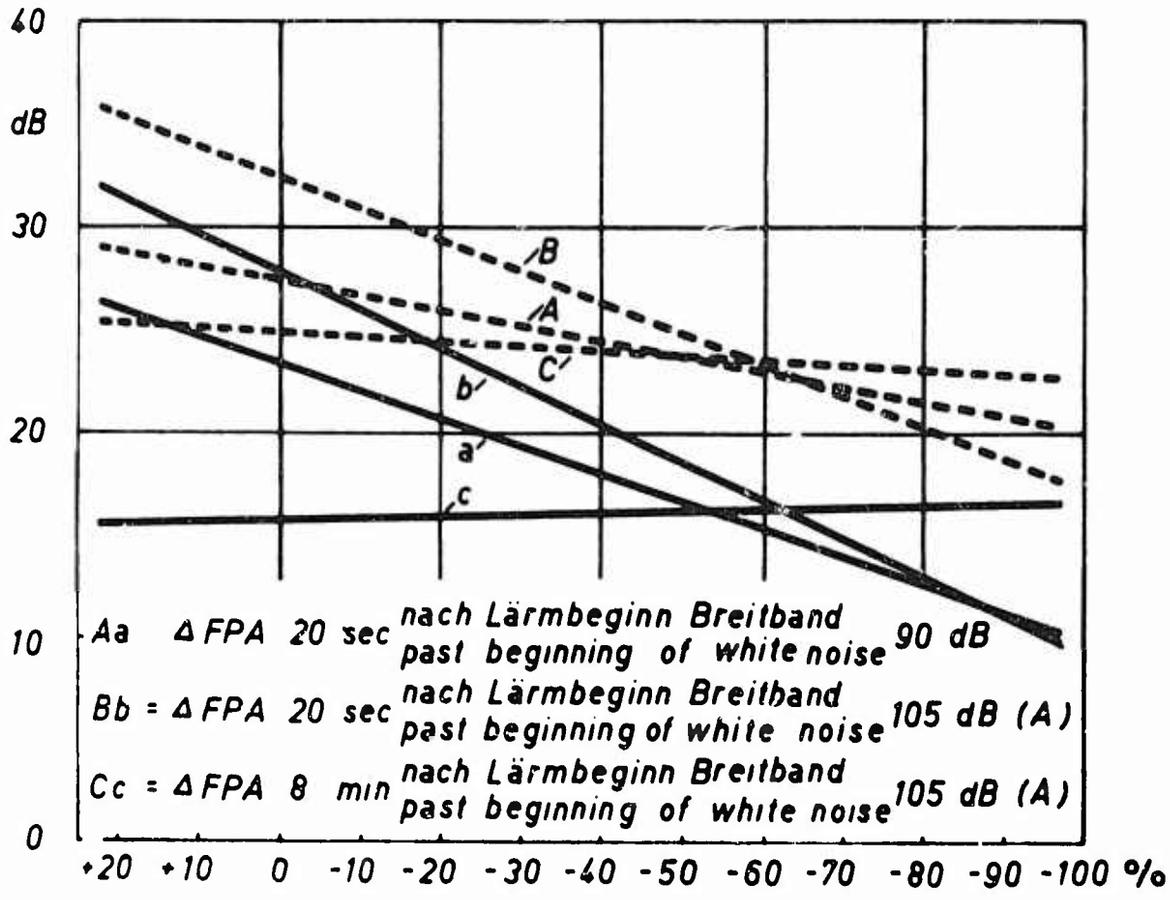

FIG. 4. Finger - pulse - amplitude and TTS. (From Jansen, G.Relation Between Temporary Threshold Shift and Peripheral Circulatory Effects of Sound. Physiol. Effects of Noise. ed. by Bruce L. and Annemarie S. Welch, Friends of Psychiatric Research, Inc., Maryland Psychiatric Research Center and The Johns Hopkins Univ. Schl. Med.,Baltimore, Md., Physiol. Effects of Noise, Plenum Press - New York - London, 1970,pp. 67-72. With permission of the authors and Plenum Press).

indicated incorrect responses on the part of the subject; its aversive effect otherwise was small.

Hormann, et al[22] in a similar study verified the aversive effects of noise with three groups of subjects exposed to white noise at 95 dB. For Group A, the noise signaled they had made an error in a pseudo-tracking task; for Group B the same noise was the signal that they were on target in the pseudo-tracking test; and for Group C the noise was heard without any task.

Measures were: (a) TTS; (b) muscle tension measured by electromyography; and (c) subjective scaling of the amount of annoyance and disturbance induced by the noise and of the general sensitivity to noise, of the subjects.

The results showed subjects who invest the noise with positive emotional valence, feel themselves less annoyed, less disturbed and, in general, less susceptible to the noise than subjects who receive the noise with negative valence.

Muscle tension was highest for Group A, less for Group B, and least for Group C.

The amount of TTS was dependent upon the valence of the noise:

1. Negative valence (Group A)TTS=18.1 dB.
2. Positive valence (Group B)TTS=12.8 dB.
3. Neutral valence (Group C)TTS=11.0 dB.

The response-contingent effect apparently does not hold for all physiological reactions to noise. Jansen and Klensch[23] found similar responses in the circulatory system (cardiac output, minute flow volume) in subjects exposed to random noise or music of equal intensity (about 90 Phon). Although the cardiac output and minute flow volume increased in some subjects and decreased in others, indicating an individual difference in

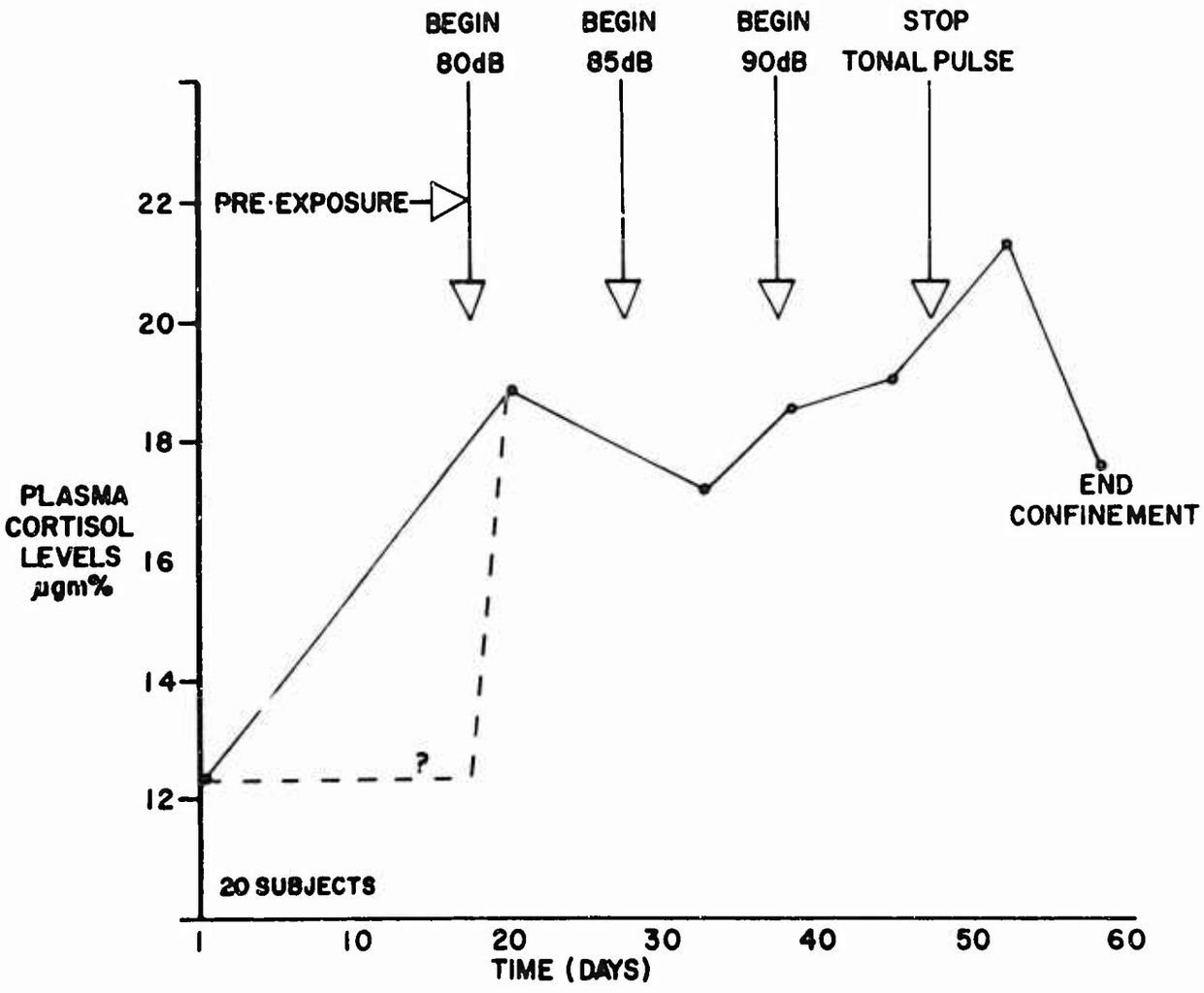

FIG. 5. Mean plasma cortisol levels before, during and after exposure to tonal pulses. A.M. sample. (From Cantrell, R.W.: Prolonged Exposure to Intermittent Noise: Audiometric, Biochemical, Motor, Psychological and Sleep Effects. The Laryngoscope, Suppl. 1,Vol.LXXXIV, No.10, Part 2, Oct. 1974. With permission).

somatic responses to sound, it was the intensity of the sound and not its aversive (noise) or pleasurable (music) aspect which controlled somatic responses.

Levi[24] measured urinary catecholamines as a method of determining N-responses in human subjects. He found the following:

1. Pleasant stimuli (motion pictures evoking amusement) were nearly as potent as unpleasant stimuli (motion pictures evoking anger) in causing increased excretion of catecholamines.

2. Work in industrial noise and office work caused increased excretion of catecholamines.

3. Noise, light, or task have less influence on the catecholamine excretion levels than does the subject's attitude.

4. Under experimental stress, emotionally vulnerable people as a group do not excrete more catecholamines than normal people.

Lehmann and Tamm[3] studied circulatory changes in human subjects exposed to noise. Peripheral arterial resistance was found to increase under the effect of noise, but pulse frequency and blood pressure remained unaffected. FIG. 1. summarizes the circulatory reactions observed by Lehmann and Tamm.

Meyer-Delius[4] relate the circulatory effects to the duration of noise exposure. An exposure of 90 dB(B) for 20 seconds increased peripheral arterial resistance for 80 seconds, i.e. the vasoconstriction mediated through the autonomic nervous system in response to noise exposure persists after the exposure.

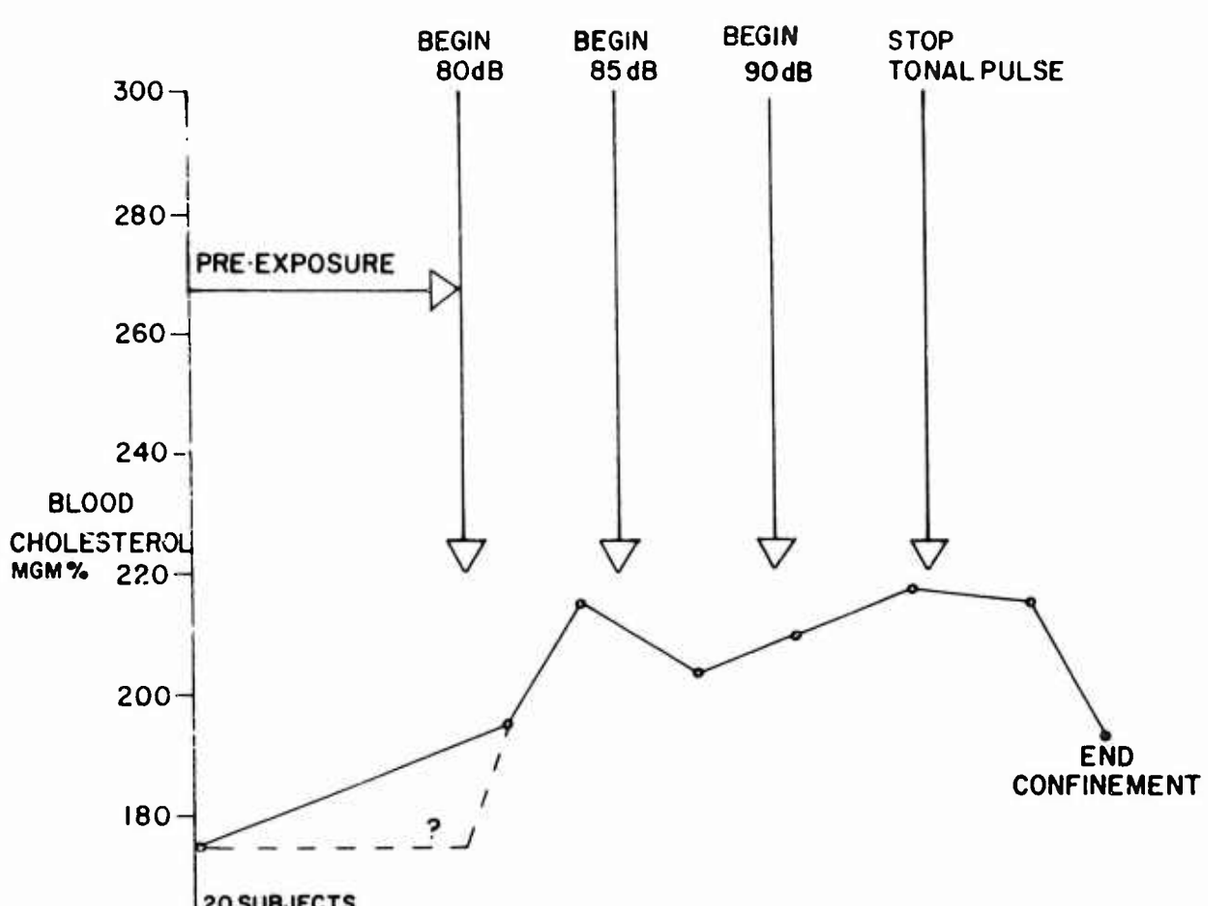

FIG. 6. Mean blood cholesterol levels. (From Cantrell, R.W.: Prolonged Exposure to Intermittent Noise: Audiometric, Biochemical, Motor, Physiological and Sleep effects. The Laryngoscope, Suppl. 1, Vol.LXXXIV, No.10, Part 2, Oct. 1974. With permission).

There is a physiological adaptation to sound. Habituation might be a more correct term, but adaptation is used more frequently in the literature when referring to this phenomena. Bartoshuk[25] found that acceleration of the heart rate in unborn babies exposed to bursts of acoustic clicks at 85 dB adapted by the end of 40 trials. This adaptation is not complete and obviously does not cover all N-responses. Although man adapts to background noise, he will respond when the character or intensity of the noise is changed. Rossi, et al[26], found adaptation of vasoconstriction in subjects exposed to a background noise of 70 dB at 500 Hz did not reduce vasoconstriction to superimposed tones of 80 to 105 dB at 2000 Hz.

Jansen[27] plotted the increased numbers of peripheral circulatory problems, heart problems and equilibrium disturbances in German industrial workers in very noisy industries. The differences in peripheral circulation and cardiac problems in the two groups were statistically significant (FIG.2).

Andriukin[5] showed a greater incidence of hypertension in men and women working in very noisy areas than their counterparts working in less noisy areas (FIG.3). There was also a relationship to age with older people appearing to exhibit more hypertension.

Additional data from Russia[6,7] has shown that among workers in industries with high noise levels there is an unusually high incidence of circulatory, digestive, metabolic, neurologic and psychiatric problems.

Rosen and co-workers[9] studied the Mabaans, a primitive tribe living in the Sudan. This tribe has no firearms or drums; their diet consists mainly of vegetables and some fish; and they live in very quiet surroundings (35-40 dB[C]) with relatively little stress. Hearing loss, hypertension and cardiovascular disease is virtually unknown even into old age.

Once the Mabaans move to noisy Kartoum, where they are exposed to noise, stress and a diet similar to city dwellers, they are reported to develop hypertension, coronary artery disease and hearing loss.

Critics of the concept that noise can cause pronounced physiological effects point out that a given noise exposure does not affect all persons similarly, nor does the same individual respond similarly to a given noise exposure occurring at a different time. These are valid observations which complicate the investigation of noise effects. A partially satisfactory answer is that no two humans are alike nor do they respond identically to any stress-stimuli. Hormann's study[22] of the valence effect of noise (i.e. a desired sound is less damaging than an unwanted sound) is an interesting concept. This may give some insight into why some people are content to work in a noxious stimulus and show little or no effects, whereas others are upset by the same noxious exposure and respond with the symptoms of stress.

A very interesting experiment is reported by Jansen[28]. He measured the change in finger pulse amplitude 20 seconds after the onset of white noise at 90 dB(A); 20 seconds after the onset of white noise at 105 dB(A); and 8 minutes after the onset of white noise at 105 dB(A). Hearing was measured before exposure and the TTS at 2 minutes after exposure ceased. The TTS was measured at 4000 Hz. FIG. 4 is a graph relating TTS and change in finger pulse amplitude (peripheral vasoconstriction). Note that for short exposures wherein you would expect little or no TTS there was rather greatly decreased FPA. With longer exposures the TTS and change in FPA were similar. Jansen concluded that the vegetative response (VR), as manifested by changes in the finger pulse amplitude, and TTS can be influenced by noise acting through the vegetative system (A.N.S.). Furthermore, a man who will not have a hearing loss from high intensity noise is, nevertheless, highly endangered by the non-auditory physiological effects of high intensity noise.

In studies of our own[10], we noted that even though 20 healthy young subjects showed little (3-5 dB) TTS after 30 days exposure to intermittent noise presented 0.66 seconds every 22 seconds 24 hours per day, they did have statistically significant shifts in plasma cortisol levels (FIG. 5) and blood cholesterol levels (FIG. 6). This noise was in the 3000-4000 Hz range and was presented at 80, 85 and 90 dB(A) each for 10 days. These are allowable levels by many damage risk criteria. This stimulus caused reduction in finger pulse amplitude during sleep and this, coupled with the relatively small TTS, supports Jansen's findings.

The shift in the blood cholesterol and plasma cortisol levels is interesting. Plasma cortisol is known to be influenced by ACTH and other studies[29,30] have suggested that stress will elevate cholesterol and cortisol. Although controversy exists as to the normal values for serum cholesterol, the range is roughly 150-300 mgm% for all ages. Younger people should normally have lower levels. In our study, the mean age was 20.7 years and accordingly, the upper limit of normal should be 189 mgm% (Keys)[31] to 240 mgm% (Fredricksen)[31]. If 189 mgm% is used as the upper limit of normal, all mean cholesterol levels during noise exposure are above normal. Even with the higher limits, all values are statistically significantly elevated from the mean, pre-exposure level, and they begin to decrease after the noise exposure ceases. In this case, the subjects acted as their own controls since all other factors including confinement, diet, and lack of exercise persisted for 10 days after the noise was stopped. Noise exposure was the only variable that changed.

These findings support the concept that the physiological effects of noise are more serious than previously supposed, and more studies of the effects of noise exposure are indicated.

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DISCUSSION

Q. (Johnson) Do you feel that noise that is so low in level that it will not damage the auditory system will cause any non-auditory effects?

A. (Cantrell) Perhaps that is a loaded question because, as you know, there is a great deal of work going on to determine a safe level of noise that will not damage the auditory system. Some people think it is 90 dBA, others think it is as low as 70 dBA. If one accepts this later statement of 70 dBA being potentially hazardous, then I think that noise under 70 dBA will not cause any physiological effects. The level of noise at which physiological effects begin, and the seriousness of these effects, is not well-enough measured yet to answer your question. My personal opinion is that somewhere between 75 and 80 dBA, both for hearing and for physiological effects, will turn out to be the critical level. I would like to have Dr. Jansen comment on this question also.

A. (Jansen) One observes vegetative reactions at very low levels of noise. It is only a question what method one uses. For example, electrodermal response, or other sensitive physiological or pyschophysiological methods, will show that there are quantitative influences at low noise levels. The question is what is the physiological relevance of these changes? I think the question cannot be answered until now as to where the point is that the normal physiological response is accumulated into a pathological one. This is the question one needs to answer. At the present one has no exact point to state where this begins. It is an increasing continuous augmentation of these reactions.

AN INVESTIGATION OF AIRCRAFT VOICE COMMUNICATION SYSTEMS AS SOURCES OF INSIDIOUS LONG-TERM ACOUSTIC HAZARDS

by
Robert T. Camp, Jr.
Ben T. Mozo
James H. Patterson, CPT, MSC
U. S. Army Aeromedical Research Laboratory
Fort Rucker, Alabama 36360
U. S. A.

SUMMARY

Military aviation operational environments contain various acoustic hazards many of which have been identified. Some sources of noise such as the aircraft voice communication systems, however, have not received much attention in terms of acoustic hazards. Recent investigations of this laboratory have shown that the acoustic output of aircraft communication systems may be an insidious long-term acoustic hazard.

The purpose of this presentation will be to quantify the acoustic output of voice communication systems in terms of magnitude and durations of exposures to these acoustic stimuli during training flights. The results of analyses of samples of aircraft voice communication systems noise will be presented.

One formidable obstacle to research on the effects of sound on the auditory systems of humans and animals is the difficulty of obtaining a precise measure of the actual acoustic input to the animals or humans under investigation. Even in controlled experiments where animals are exposed to controlled noise environments, often it is difficult to know what the animals receive due to the gradients within the noise environments and due to the orientation of the head and the pinna. Some investigators have spent years in the development of techniques and methods for measuring sound pressure levels in human external auditory canals.

In hearing conservation programs and studies of the effect of aircraft noise on the aircrewmen the translation of the sound pressure level values measured in the acoustical environment into the actual acoustic stimuli of the people operating in the environment is no less difficult. The trend in the last decade of using overall dBA measures of sound pressure levels in various military and industrial environments has perhaps spread some measure of confidence among the laymen that all one needs to do is to take a sound level meter survey of a working environment to predict the acoustic hazards in which personnel work. Our investigation of the acoustical environment of aircraft crewmen has led us to the philosophy that a true knowledge of the actual acoustic environment requires a continuous refinement of instrumentation and knowledge of the physical stimuli and a careful consideration of all factors that might affect or constitute the total acoustic input into the ears of the crewmen under study. This presentation is about the results of some of our recent investigations which show how the acoustical environment measures of aircraft may be much more than a casual assessment with ambient sound pressure level readings taken with sound level meters. My subject is: "An Investigation of Aircraft Voice Communication Systems as Sources of Insidious Long-Term Acoustic Hazards."

We have heard from other presentations about the acoustic environment of patrol aircraft crewmen and a report on the hearing loss statistics along with the sound pressure levels associated with the operation of these aircraft. In the photographs, the crewmen were wearing headsets which is normal for operations in these aircraft. The problem in relating hearing loss statistics and the associated alleged acoustic environment is that the aircraft have great variance of sound pressure levels inside the various compartments due to a multitude of noise sources, gradients and reflections. It is difficult to know the exact positions and the durations one operates in the aircraft. Another problem in assessing the true acoustic input to a member of a crew is due to the headset which may cover his ears. Headsets are generally efficient attenuators at high frequencies and usually have little attenuation in the low frequency range. But even with this knowledge, one cannot derive precisely what the total acoustic input is to the crewmember, for during training or flight operations he is continuously receiving communication signals from the intercom system and radio transmission mixed with high sound pressure level system noise.

In other types of aircraft, such as U. S. Army helicopters, cockpit and passenger compartment noise, sound pressure levels are usually very high and constitute a damage-risk to hearing. Since the development of the SPH-4 helmet the problem of damage-risk to hearing by U. S. Army aircraft engine and rotor noise has been reduced, for it has been determined that the effective sound pressure levels, at the ears of crewmen wearing the SPH-4 helmet, of these noises are below 85 dBA. The attenuation characteristics of SPH-4 helmets provide protection even in the heavy-lift helicopters with extremely high noise levels, and also the helmets protect against most of the extremely high level weapons impulsive noises that are generated in and around helicopters. The investigation of the efficiency of the helmet with the use of a small microphone mounted inside the crewmen's ears has furnished data that show that the SPH-4 helmets do provide protection as was calculated with the known noise spectra in the aircraft and the known

real-ear attenuation characteristics of the helmets. The measurements beneath the helmet during training flights have shown that our predictions of effective engine and rotor noise levels are correct, but also revealed that the aircrewmen are not entirely isolated from high sound pressure levels. It was found that the radio equipment coupled directly to the ears of the pilots is a source of sound pressure levels much higher than the effective level of the engine noise when the helmet is worn.

Measurements of the duration of sound pressure levels in a CH-47C helicopter at the pilot's ear without helmet are shown in Figures 1 and 2.

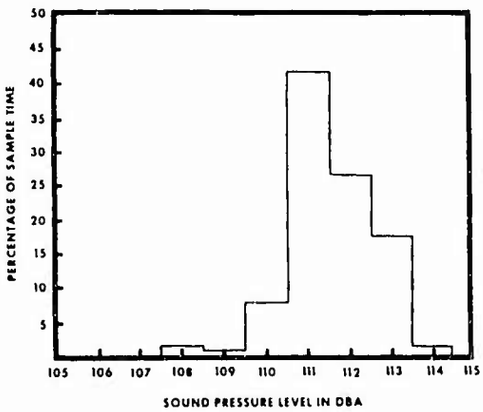

Figure 1.

Duration of Sound Pressure Levels in Percentage of Sample Time Measured in a CH-47C at the Pilot's Ear Without the Helmet

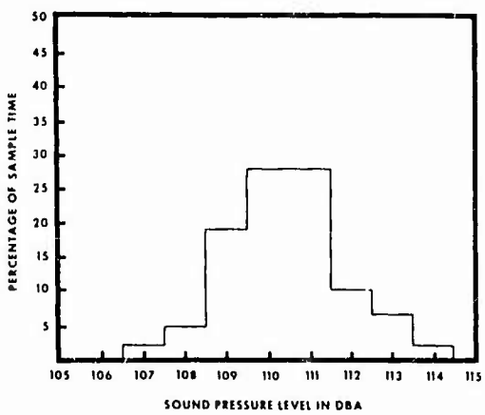

Figure 2.

Duration of Sound Pressure Levels in Percentage of Sample Time Measured in a CH-47C at the Pilot's Ear Without the Helmet

These figures contain data from samples in two different CH-47C's. The mode of the distributions of sound pressure levels in Figure 1 is 111 dBA, and in Figure 2 the mode is between 110 and 111 dBA. These data show the effective level at the ears of the pilots when no helmet is worn during the operation of the CH-47C helicopter.

Figures 3 and 4 contain duration of sound pressure level distribution in terms of percentage of sample time measured in a CH-47C at the pilots' ears with the helmet on and without communication system operating.

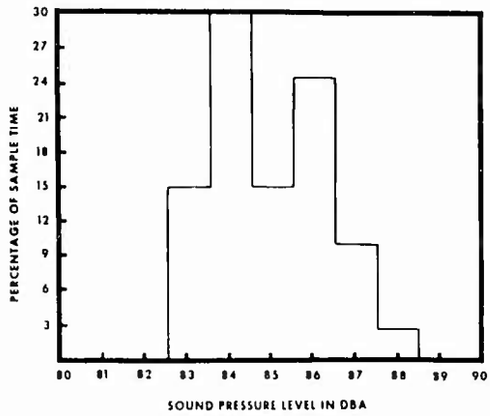

Figure 3.

Duration of Sound Pressure Levels in Percentage of Sample Time Measured in a CH-47C at the Pilot's Ear with the Helmet On and Disconnected from the Communication System.

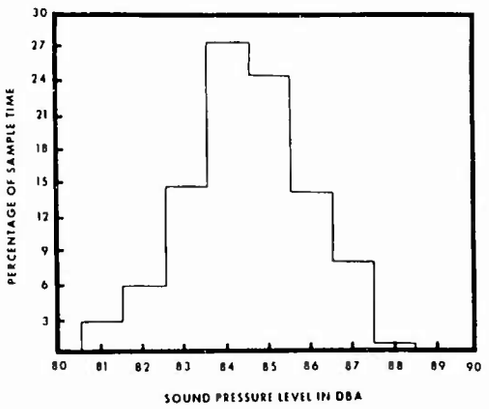

Figure 4.

Duration of Sound Pressure Levels in Percentage of Sample Time Measured in a CH-47C at the Pilot's Ear with the Helmet On and Disconnected from the Communication System.

It may be noted that the modes in Figures 3 and 4 are 84 dBA.

The effect of the operation of the communication system on the acoustic input to pilots' ears is shown in Figures 5 and 6.

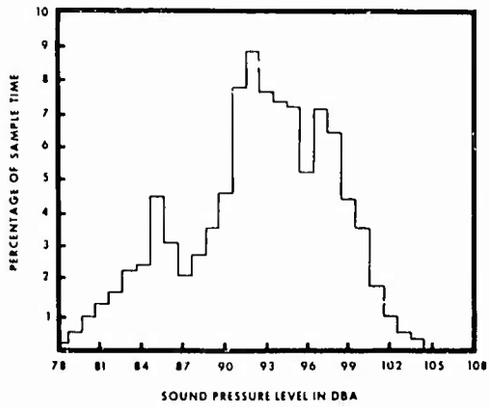

Figure 5.

Duration of Sound Pressure Levels in Percentage of Sample Time Measured in a CH-47C at the Pilot's Ear with the Helmet on and Normal Communication System Operation.

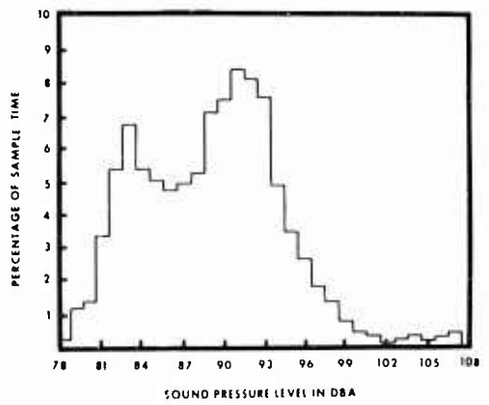

Figure 6.

Duration of Sound Pressure Levels in Percentage of Sample Time Measured in a CH-47C at the Pilot's Ear with the Helmet on and Normal Communication System Operation.

These distributions contain the duration of sound pressure levels in percentage of sample time measured in a CH-47C at the pilot's ear with the helmet on and with communication system operating intermittently. One would expect the obtained bimodal distributions in view of the high sound pressure levels emitted by the two communication systems and the effective levels at the ear when no communication system is operating. The modal value of the aircraft noise, at the ears while wearing the helmet, was 85 dBA, and other modes in the two figures ranged from 91 up to as high as 96 dBA when the voice communications system was operating. It is therefore apparent that the communication system produces as much as 10 dB higher sound pressure level than the effective sound pressure levels of the aircraft engine noise.

After it was determined that the voice communication systems do produce high sound pressure level acoustic outputs, it was speculated that perhaps the distortion and the low quality of the systems might affect the level at which pilots set the sidetone during flight operations. A pilot experiment was conducted to investigate how the sidetone level was affected by various amounts of peak-clipping for the 'best understanding.' Multiple-choice intelligibility words were presented under four conditions. One condition had no distortion, the second condition had 1 dB peak-clipping, the third condition had 3 dB peak-clipping, and the fourth condition was with 6 dB peak-clipping.

In Figure 7 it is obvious that with speech alone the amount of peak-clipping had little effect on the adjusted level. However, with speech mixed with noise - which simulates the actual conditions in aircraft - the clipping caused an increased adjusted level of the sidetone.

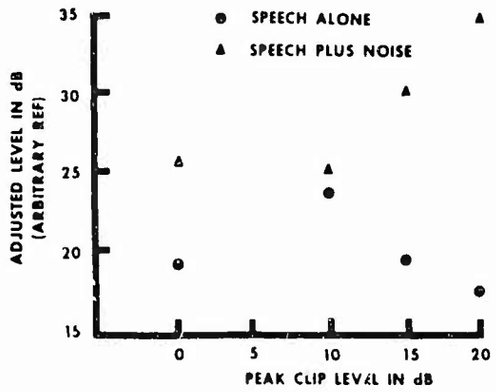

Figure 7.

Mean Level Adjusted by Four Pilots for Subjective "Best Understanding" as a Function of Amount of Clipping in dB.

A second measure of the performance of the four aviators was done in terms of the actual intelligibility of the words as a function of adjusted level. Figure 8 depicts the results in terms of intelligibility in percent as a function of adjusted level. These data show that intelligibility was highest with the lower adjusted levels and that intelligibility decreased with the higher adjusted levels. In other words, the aviators' attempt to adjust at the "best listening level" did not necessarily yield higher intelligibility scores.

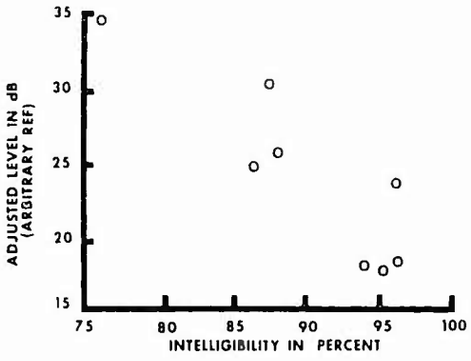

Figure 8.

Mean Level Adjusted by Four Pilots for Subjective "Best Understanding" as a Function of Percent Intelligibility.

In summary, we have presented data to show that the SPH-4 helmet is an efficient attenuator of the cabin and passenger compartment noise in aircraft. Our investigations of the efficiency of the helmet to confirm our calculated values have led us to the problem of high pressure levels generated by voice communication systems. The output from these systems may be higher than the effective level of the engine noise and may be an insidious hazard to hearing over a long period of time. Our investigation also shows that distortion may cause the high level adjustments by pilots. Theoretically, they could

adjust levels much lower than those depicted in these data. But the tendency is to set levels higher than necessary when distortion is introduced.

It is therefore recommended that future communication systems be designed without distortion in order to yield higher intelligibility and speech communications efficiency, and also - to eliminate the acoustic hazards that high sound pressure levels may cause over a long period of time.

DISCUSSION

Q. (Thorne) Can you indicate why you think peak clipping will reduce speech intelligibility?

A. (Camp) As a matter of fact, peak clipping will not reduce speech intelligibility very much with normal speech if it is not mixed with noise. However, under noisy conditions, such as those of the NATO mission where pilots are attempting to understand speech in noise, clipping causes extra harmonics that are useless energy and also causes masking which obscures the speech. This makes it difficult for the listener as you heard in the examples I gave If you recall, first you heard speech without noise, and then mixed with noise, and then peech that was clipped without noise, and with noise, and you could tell the difference. The intelligibility of the clipped speech was deteriorated over that of the non-clipped.

Physiological Responses Due to Noise in Inhabitants around Munich Airport

Prof.Dr.med.Dr.phil. Gerd Jansen
Universitätsklinikum Essen
43 Essen 1, Hufelandstr.55
Federal Republic of Germany

Summary: The Deutsche Forschungsgemeinschaft (DFG) initiated and sponsored an interdisciplinary research on aircraft noise effects on inhabitants around airports. A pilot study (around Hamburg airport) and a main study (around Munich airport) were conducted by acoustical, demographic, social scientific, psychological, physiological and medical sections of scientists. It was found out that, in general, there was no adaptation to aircraft noise. There is existing a linear relation between increasing noise stimuli (combined noise exposure measure of noise levels and number of flyovers) and human reactions esp. found in social scientific fields. The discussion of physiological results leads to the opinion that physiological reactions are more related to sound levels whereas the "whole reaction" (annoyance, blood pressure etc.) is more related to combined noise exposure measures.

1. Scope

The effects of aircraft noise on men living around airports were studied by an interdisciplinary team which was sponsored by the Deutsche Forschungsgemeinschaft (DFG). The main study was conducted around Munich airport and the pilot study was done around Hamburg airport.

There are existing some other 15 investigations in the world using survey technics but they confined to mainly sociological aspects such as complaints or annoyance. Thus DFG initiated than an interdisciplinary research including not only sociological and psychological, but also physiological impact of aircraft noise.

2. Organization of the study

The team of the project was composed of 6 sections:

- "Acoustics" (H.-O. Finke, R. Martin; PTB Braunschweig)
- "Medicin" (A.W. v. Eiff, L. Horbach, H. Jörgens; Uniklinik Bonn)
- "Organization" (B. Rohrmann; Uni Mannheim)
- "Psychology" (R. Guski, H. Hörmann; Uni Berlin, Uni Bochum)
- "Social-science" (M. Irle, R. Schümer, A. Schümer-Kohrs; Uni Mannheim)
- "Work-physiology" (G. Jansen, Uniklinik Essen)

Each section tested the same subjects to collect data for an interdisciplinary analysis.

Preparing the whole study the organizational section first selected the human beings living around Munich airport according to the exposure to the noise levels and secondly in accordance with demographic criteria. The whole area was divided into 32 areas with different noise levels; these "clusters" were combined to 4 "cluster-sets".

660 persons from 15 to 70 years were tested in a first social scientific step. Interviews based upon standardized questionnaires were taken at the respondents homes. This interview had a contacting function in order to ask the people to follow the second step of our investigation in psychological, physiological and audiometric experiments and tests. The examinations of 357 subjects in a separate test station took about 2 hours for each person.

The third step contained the medical case history, clinical examination and experiments at the test station. This step took another 2 hours for each person. The fourth step was a concommitant one, it consisted of acoustical measurements (one measuring point for each cluster). The tested subjects in the first and in the second step (psychophysiological and medical examinations) were restricted to 375 persons aging from 21 to 60 years.

The interviews of the sociological section were extended in a second part to 152 former inhabitants of the clusters who had moved during the last 12 months preceding the study.

3. Results

3.1. Former publications

The major results of the whole DFG-study were already reported at the congress at Dubrovnik in May 1973. Another survey of the results was given at Inter-Noise (Copenhagen 1973). Especially the relations between acoustic parameters and noise reactions in human beings were presented at the Transportation Noise Symposion (Southampton, July 22-23, 1974). The detailed DFG Forschungsbericht is in print and publication is expected on February 15, 1975.

3.2. Main results of the whole study

According to the assumption of the complex (multicausal instead of monocausal) system of interdependent variables these "moderator variables" were being attributed and regarded as decisive influences on the process of turning effecting stimuli into resulting reactions. This concept of moderators led to an interdisciplinary analysis and synthesis based on an analysis of the single sections. Thus, it is useful to know first the results of the single sections.

3.2.1 Social survey

By means of regression and correlation technics the social science section tried to clarify the relationship between stimulus moderators and reaction and between the relative contribution of stimulus and moderator variables to the prediction of reactions. It was found out that the relationship between stimulus and reaction variables are by no means perfect ones; the highest correlations ranked to r 0.56.

This result means that only about 30 % of the variability in reactions can be predicted by means of one stimulus variable alone so that a considerable amount of the whole reaction remains unpredicted.

As in other aircraft noise studies "disturbance of communication (disturbance in conversation, in listening to radio, TV)" was the greatest one, whereas other ones like "perceived number of times of aircraft noise", "irritability by aircraft noise","disturbance of tranquillity and relaxation", "the number of subjects spontaneously naming aircraft noise when asked for interconveniences" etc. they all had smaller correlations.

All these relations are linear. Curvilinear determination coefficients led only to an insignificant increase as compared to linear determination coefficients. Even when correlating more than one stimulus variable no other result could be found.

3.2.2 Psychophysiological experiments

The psychophysiological laboratory experiments were done by the psychologists and work-physiologists together. On one side we had the hypothesis of "adaptive coping" with aircraft noise. On the contrary we tried to find out a "defensive blocking" which assumes an interruption of information processing and physiological state of defensive against noise, as a consequence of frequent and intense aircraft noise. Therefore the investigation was done under the aspect of the "general activation theory" and its possible splitting into "orienting" and "defensive" components.

Moreover it was assumed that damping or disturbance of the information input is in accordance to the "distraction theory"; further on it was expected, there were connections between aircraft noise stimuli and aircraft noise reaction by personality characteristics.

In order to find out the characteristical noise reaction of the people investigated, the psychologists used personality tests, recognition technics, memory tests, signal tracking test, and together with the physiological reaction they registered the behaviour of vasomotoric and muscular activity which were continuously recorded in experimental situations with quietness and noise interchanging.

It was not possible to confirm the hypothesis of "adaptive coping" with aircraft noise. The physiological responses due to noise increased in all cases. In detail, we saw a contraction of the bloodvessels at the finger and at the temple, an increase in the electrical muscle activity, and a decrease of the heart rate. This complex reaction was called "defensive reaction" following SOKOLOFF. One can conclude from this that there could be at least a blocking of information reception processes. These defensive reaction is correlated positively with the intensity and frequency of the aircraft ($r = 0{,}21$). It occurs especially with those persons who were characterized by a "low mobility", by "strong conservative tendencies" and by a "very high blood pressure".

Moreover we saw that the hearing acuity decreased with increasing aircraft noise exposure. But this result is statistically insignificant. Other respects of human psychophysiological behaviour especially psychological behaviour were not so much effected by aircraft noise.

3.2.3 Medical investigation

The medical examinations were done separately from that of psychophysiological sections after another two weeks. The people were assessed by means of anamnesis and examination of body containing the analysis of clinical status as well as experimental tests of vegetative functions.

The analysis of the medical data could not prove any cause of manifest illness which is due to aircraft noise. In physiological experiments systolic and diastolic blood pressure, heart rate, respiration rate and electrical muscle activity were recorded for 15 minutes. The subjects were submitted to quietness, mental arithmetics, continuous noise, and discontinuous noise. There was only a tendency of change in vegetative functions especially regarding the diastolic blood pressure. The medical scientists have the opinion that it cannot be excluded that aircraft noise is a "risk factor" for the generation of essential hypertonicity of the bloodvessels.

3.3. Interdisciplinary interpretations

The different data from the single sections were integrated (N = 357) to an interdisciplinary analysis which resulted only in low intercorrelations of the sociological, psychological, and physiological variables towards aircraft noise effects.

Using an interdisciplinary set of sociological, psychological and physiological moderators 1/3 of the variability of the "social-psychological" is determined by them whereas another third is determined by the stimulus variables. By using so called path models the scientists doing the interdisciplinary interpretations found chains within one path model containing the factors "indifference to noise", "age", "sex","fear associations" "attention performance" connecting them with "annoyance and disturbance reaction", "defensive reaction", "diastolic blood pressure" and the dependence of all of them to the noise load.

With reference to the noise protection zones as they are defined in several countries like USA, Great Britain, West Germany, etc, the scientists doing the interdisciplinary analysis found out that outside of the areas confined by this protection values there is a considerable percentage of the population which is highly annoyed and influenced by aircraft noise.

But the regression lines of the "disturbance of communication", "disturbance of rest and recreation" and the "feeling of aircraft as a disturbing factor spontaneously mentioned" were linear regression lines. So there is no point which could be regarded as intolerable noise load. There is only an increasing number of people who feel annoyed and who are influenced physiologically by increasing of aircraft noise. So they conclude that the reduction of aircraft noise is a problem for those producing noise and also for those distributing noise. They feel that it is a problem involving aspects of engineering as well as of policy.

4. Discussion of results from physiological standpoint

Already the pilot study around Hamburg airport showed and proved that the results of former physiological noise research need no basic correction. The experimental physiology results of the Hamburg pilot study (aircraft noise, traffic noise and artificial white noise were applied), showed that the results were comparable to those expected from results of former noise research.

In the main study around Munich airport we tried to find out moderating factors of the physiological responses. These could give explanation of the value of the psychophysiological noise reaction within the total load of environmental factors of the human being. We stated already that the theory of "adaptive coping" had to be cancelled in favour of the "defensive reaction". The combined defensive reaction consisting of changes in finger and headskin blood volume, muscle activity and tracking test, were regularly influenced by single noise bursts. Though the whole defensive reaction is correlated in a linear regression to the combined noise measure FB1 (which contains the number of movements and the noise levels of the single movements similar to the English NNI (noise number index))we saw the most distinctive reaction in the finger pulse amplitudes. Comparing these results with former investigations done with approaching aircraft noise and with the noise reactions of people with different personality moderator variables we think that the physiological measuring parameters are closer correlated to noise intensity level whereas the combined reaction in the physical as well as in the psychical behaviour is more related to the combined noise exposure measure (number of movements and intensity level).

This leads us to the conclusion that for noise assessments around airports it is necessary to have first a combined measurement unit (as they used already internationally) and second (for realistic assessment and protection of the population) to have a maximum level for single noise events.

DISCUSSION

Q. (von Gierke) I agree with Dr. Jansen's statement that we have physiological responses to noise. We also heard in Dr. Cantrell's review about all of the physiological responses to noise which most of us think are very healthy and natural responses to our environment. Unless we have evidence that some of these transient physiological responses become chronic or somehow lead to chronic diseases and pathological effects it is really nothing to worry about. In all the research that I have followed over the past twenty years I have not been able to come up with any clear cut proof that there is a chronic health effect from the levels of noise exposure that we are talking about. I am not saying that these effects don't exist, but the only study which shows such a correlation was Dr. Jansen's study, which was cited before, on noise in industry. This was done 15 or 20 years ago and hasn't been replicated since. This study showed a potential indication that noise exposure in industry might be correlated with some increase in cardiovascular disease. However, the same workers in noise that were studied had many other environmental factors associated with their work which might have been just as bad if not worse than the noise itself. Studies have been performed recently on mice and rats that show pathological effects to high level noises but I think we should really concentrate on studies in the real life situation that are made on man rather than on mice and rats. The stories we hear about malformations and reduced fertility in litters of rats and mice are open to some question. First, the noise levels are high. Second, it happens that there are not good controls used. When controls are handled the same way the experimental animals are handled these effects diminish.

A. (Jansen) You mentioned my study of twenty years ago. Yes, it should be replicated and we are just now undertaking experiments in order to find out the relevance of noise along with other factors in producing health effects on workers. We have a group of young men who are doing their research thesis just on this point. Perhaps it is possible within one or two years that we will have the results that you were asking about.

A. (Cantrell) I would like to answer Dr. von Gierke regarding the statement he just made. I hope that from the presentation I gave one didn't infer that there was an attempt to offer any clear-cut proof that there is a patho-physiological effect of noise, but rather that the indication is clear. It is clear in animals. Although we cannot necessarily apply animal studies to humans, nonetheless we do have the human studies of Dr. Jansen as well as studies done in Russia and Europe. Unfortunately, few studies have been done in the U. S. Part of my presentation was a plea for more activity in this problem area. I do feel, however, that prolonged exposure to noise, noise meaning unwanted sound, must act as a stressful stimulus. Theories of the effects of accumulated stress have been present for over forty years. Most people now agree that stress is a factor which causes followon patho-physiological problems. The stress of noise, if noise does cause stress, and I think it does, can very well lead to patho-physiological effects. We should study this. Whether we can ever say for certain that noise exposure for a given period of time at a given level is going to cause hear' disease or biochemical abnormalities of certain kinds is not likely, but I think that certainly further studies need to be performed and that we agree on this point.

A. (von Gierke) I think it is simpler just to say that noise affects the quality of life and what we want to do is improve the quality of life.

A. (Ward) I disagree with Dr. von Gierke. Improving the quality of life is only one of the things we are after. More importantly, we are interested in the effects of noise on health. Not health as defined by the World Health Organization which includes such things as feelings of well-being. Let's talk about health as absence of pathology. True, we are interested in protecting the public's feeling of well-being in the long run but first let's concentrate on protecting the public from pathology.

A. (von Gierke) Noise is a stress, I agree. But we are exposed to many stresses during the day. Sitting on these chairs for eight hours is a stress and it just depends on how great the stress is. In all seriousness we once tried to follow-up some of Dr. Jansen's work and that of some other workers by obtaining vasoconstrictive responses to vibration stimuli. We exposed the arms of our subjects to vibration. We worked for hours, even days, until we had a nice response of vasoconstriction resulting from localized vibratory stimuli on the skin. We had this effect, finally, and suddenly the pointer went completely off the scale. It turned out that a young woman had walked through the room and our male subject was so stressed that the vaso-constriction from this stimulus was far more violent than from the laboratory vibratory stimulus.

Q. (Olson) How many people moved away from the Munich airport because the noise irritated them?

A. Those who moved did not do so because of noise nor were they found to be more sensitive to noise than was a control group.

Q. (Perdriel) I waited until all the papers were presented before asking any questions because I thought that one of the speakers might discuss the effect of noise on the visual system. For a number of years it is well known that exposure to high intensity noise for several minutes or for several hours can bring about changes in the visual function and thus might endanger flying safety. We have studied the effects of noise of 5,000 Hz, or a complex of 5,000 Hz, the intensity of which ranged between 85 and 105 dB, on the parameters of the visual system and we have found a decrease of about 20 % in the night vision appa- [illegible] during exposure to noise. This was determined by measuring the [illegible] night thres- [illegible] holds. These are [illegible] values [illegible] were [illegible] per- [illegible] were slightly [illegible] was [illegible] [illegible] per- [illegible] [illegible]

important to find out the anatomical location responsible for these interactions. We believe that the thalamus may be the responsible site for interaction between the auditory and visual functions. In fact an afferent activity of the sensory visual and auditory processes (pathways) does take place in the thalamus. Based on electro-corticoid studies of the thalamus it may be assumed that an interference disorder) occurs at this site leading to a diminished passage of the sensory visual messages when noise stimuli traverse the thalamus. Moreover, an inverse study carried out in Italy confirms to a degree these findings. If one exposes the eye to a strong light for several minutes one finds a decrease in the auditory threshold. This proves again an interaction between the auditory and visual sensory messages.

A. There have been many experiments performed in these areas. We have done work in our own laboratory on these problems. I think that it is not justified to generalize from these experiments to the real life situation, for one must consider man has motivation and has capability to compensate and these parameters must be taken into account. What one needs to do is to make experiments under real life conditions rather than laboratory conditions. From the experimental situation we know many things, but it is very dangerous to generalize from experiments to the real life situation.

CONCLUDING REMARKS

by

Dr. Milton A. Whitcomb
National Academy of Sciences
Washington, D. C. 20418, U.S.

It is impossible to summarize adequately, in the sense of several synopses, such an excellent set of papers as have been presented. Rather, it might be desirable to present instead a few reflections upon today's program.

There can be no doubt that noise exposures of durations greater than eight hours present a hazard to the hearing of air crews flying noisy aircraft and, particularly, for those more susceptable crew members. We were shown today that there are wide individual differences in human response to high-level, long-duration noise. The effects of long-duration exposures on performance and health are less clear. Monitoring audiometry and ear protection is certainly indicated for preservation of hearing and, perhaps, for insuring performance and ultimate health of air crews flying long-duration missions. It is of additional importance that noise reduction at the source be accomplished wherever possible for safeguarding the hearing and health of those who live around NATO airports.

Collection of data on incidence of stress-induced pathologies such as ulcers or emotional disorders for those exposed to long-duration noise, as compared to non-noise exposed might be worthwhile in order to resolve the question of whether or not health is affected. It is therefore recommended first that flight crews exposed to such long durations of noise be monitored audiometrically and for abnormal incidence of cardiovascular disease, ulcers, and other psychosomatic complaints and that secondly, if possible, a study of an appropriate laboratory animal might be instituted over the next several years which could resolve perhaps the important problem of whether or not pathology can be induced because of long-duration noise exposure to the moderate levels of noise that occur in aircraft cockpits. The following research design might be a starting point for a study to be funded through the NATO system

RESEARCH DESIGN: EFFECTS OF CHRONIC EXPOSURE TO LOW LEVEL NOISE UPON HUMAN HEALTH*

Studies have been conducted over the past twenty years that indicate a possibility of chronic health effects in humans caused by moderate noise levels (70-90 dBA) when their duration extends over a major part of one's lifetime. This possibility is suggested by three kinds of studies, none of which positively links moderate noise levels to human health. However, these studies are suggestive and ought to be validated by careful research techniques. If the results are negative one would feel a sense of relief. If positive, then steps must be taken quickly to protect the public from these health effects since the hazardous noise level would then be considerably below present levels that are based mainly upon protection of hearing.

The first line of research deals primarily with West-European studies of humans in an industrial setting and links non-auditory health effects with exposure to high levels of noise. Unfortunately, these studies are merely suggestive since high levels of noise were studied rather than moderate levels. Also, the deterioration in the health of the worker was not only linked to progression of years of exposure to these high levels but was confounded with increases in the age of workers and in their exposure to other stressors such as pollutants in the air and excessive temperatures.

The second line of research involves the exposure, primarily of rodents, to rather high noise levels. These studies indicated pronounced health effects such as enlargement of endocrine glands, loss of fertility, birth defects and genetic changes. These findings, though of great interest, again are only suggestive since the noise levels were high and the animal selected for research is not particularly good for generalization to human health effects.

The third line of research involves the laboratory exposure of humans to moderate levels of noise for brief periods of time. Results indicate, for example, such effects as peripheral vasoconstriction, temporary shifts in heart rate, blood pressure and blood chemistry. To some extent, these changes gradually approach the pre-exposure baseline as the exposure continues. A question remains concerning whether the cumulative effect of the initial change could impair one's health.

Since the question is obviously an important one to the health of NATO pilots, the following research design is suggested:

Subject	Primates or intelligent mammals
Levels	80 - 85 - 90 - 95 dBA
Spectra:	Those spectra typical of jet and reciprocating engine aircraft cockpits
Temporal Pattern	Continuous and intermittent

*Health, as used here, refers to pathology. This excludes such things as discomfort or annoyance in contrast to the World Health Organization's definition of health.

The animals should be exposed throughout their life span to a constant 24 hour-day, noise level, spectrum, and temporal pattern while having periodical biochemical assays of blood, urine, etc. as well as a careful postmortem wherein any enlargement of endocrine glands and other abnormalities can be determined. Each noise condition should be run on a sample of at least 20 animals caged in a reverberant room independent from the room housing the animals exposed to other levels and spectra. The control group of perhaps 40 animals should be housed in a relatively quiet room of approximately 50 dBA level. The temperature, humidity and physical features of the control and experimental rooms should be as identical as possible. The noise producing equipment in each experimental room should be engineered in such a way that it can produce accurate levels and spectra continuously for several years and be amenable to quick repair so that malfunctions will result in quiet periods having durations of no more than a few hours.

The parent animals should be examined prior to exposure to identify those having any ear pathology requiring their elimination from the study. Behavioral threshold testing would also be advisable at this stage for the same reason. Offspring born during the experiment should also be examined and given threshold tests as soon as they can be performed validly. Exclusion, for these reasons, of offspring from the experiment, however, is not advisable since these cases may be of interest. These data should be analyzed independently of those of healthy animals.

To perform the biochemistry and autopsies competent academic and medical personnel should be involved such as a recognized university and/or medical school in conjunction with an appropriate laboratory having competent noise experts.

The funding level may require $2,000,000 per year for several years.

The papers presented leave little doubt that long-duration noise exposures not only permit less recovery time before the next exposure period occurs within the 24-hour cycle, but of more concern is the fact that these recovery curves are less rapid than one would like and the recovery seems more resistant following the reaching of an asymptotic level of temporary threshold shift. This leads to the conjecture that long-duration noise exposures may be considerably more hazardous than those of short duration. It appears that asymptotic threshold shift occurs for humans somewhere between eight and twelve hours exposure and that if sixteen hours of exposure would occur, the recovery may not be complete following just eight hours and the flight crew would be starting off the next day with a residual temporary threshold shift to be added to by the exposures from the next day's duty. This is of sufficient concern that NATO should initiate studies to investigate this possibility while simultaneously monitoring such crews carefully. Alternatively mission cycles could be reduced to exposures of eight hours or less per day.